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POLYPLOID AFRICAN VIOLET

(SEE PAGE 225)

Mendel's Autobiography

Black-Spotting in Swine

Greetings to W. E. Castle on His Eighty-Seventh Birthday

VOLUME 45 SEPTEMBER-OCTOBER, 1954 NUMBER 5

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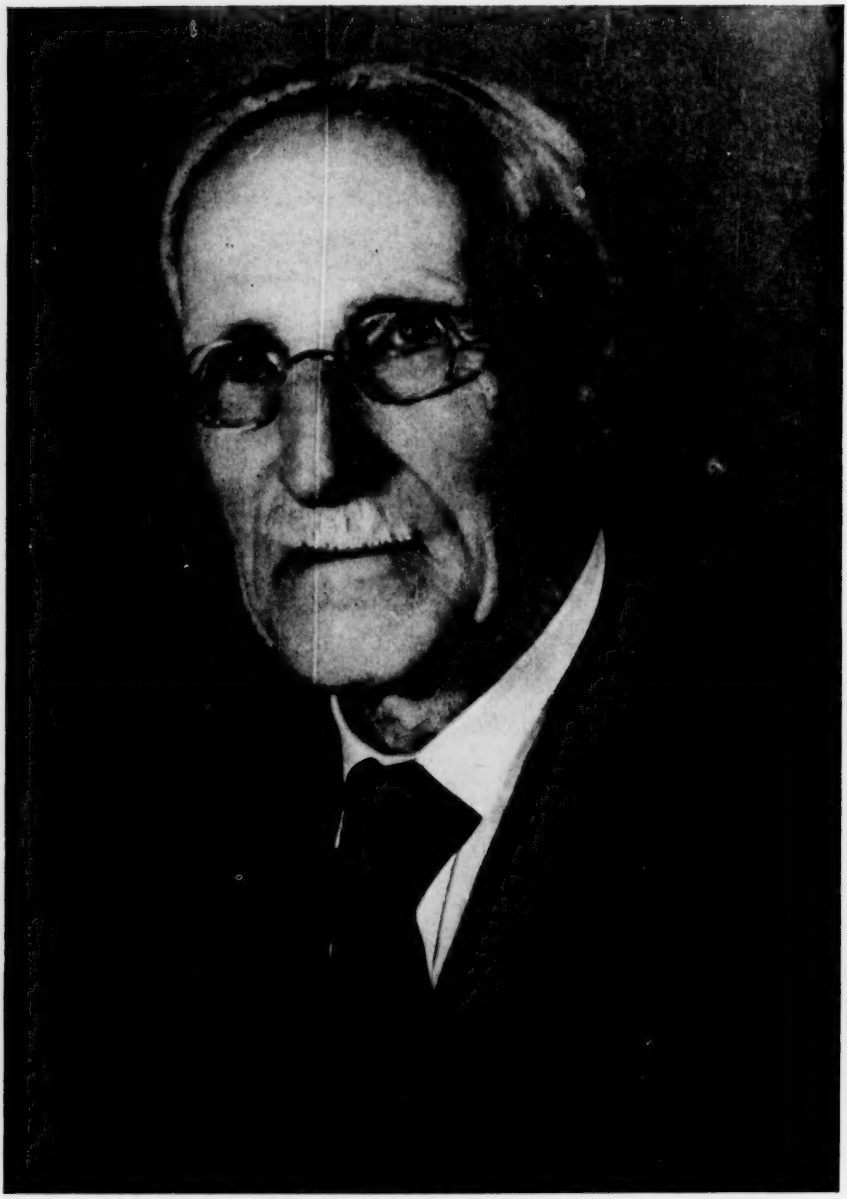


NESTOR AND INTELLECTUAL GRANDFATHER OF MAMMALIAN GENETICS

Dr. Castle, 87 on October 25, 1954, is versatile: Teacher of Latin (1889-1893), of vertebrate anatomy, of history, of zoology, and of genetics since even before the rediscovery of Mendel. A charter member of the American Breeders' Association, Dr. Castle has been an officer of the American Genetic Association since it was organized.

ONE MAN'S INFLUENCE: A TRIBUTE TO WILLIAM ERNEST CASTLE

ELIZABETH S. RUSSELL*

THE total scientific influence of a particular individual is not an easy thing to measure, since influence is a subtle and variable entity. One standard is, of course, magnitude and quality of publication, and on this basis Dr. Castle has a splendid record. But the written word certainly is not the only means of producing an effect. Many scientists feel that personal contact, shared work, encouragement, and exchange of ideas, may be even more important. Apparently an extremely favorable environment for the development of geneticists was provided at the Bussey Institute of Harvard University from the day of its opening in 1909. All of us at the Jackson Laboratory feel deeply indebted to William Ernest Castle, dean of the American Mammalian Geneticists, Professor Emeritus of Harvard University, Research Consultant at the University of California, and head of the Bussey Institute from its inception until 1937. We would like to honor him on his eighty-seventh birthday by trying to demonstrate some of the effects which this truly remarkable man has had upon the development of the science of genetics.

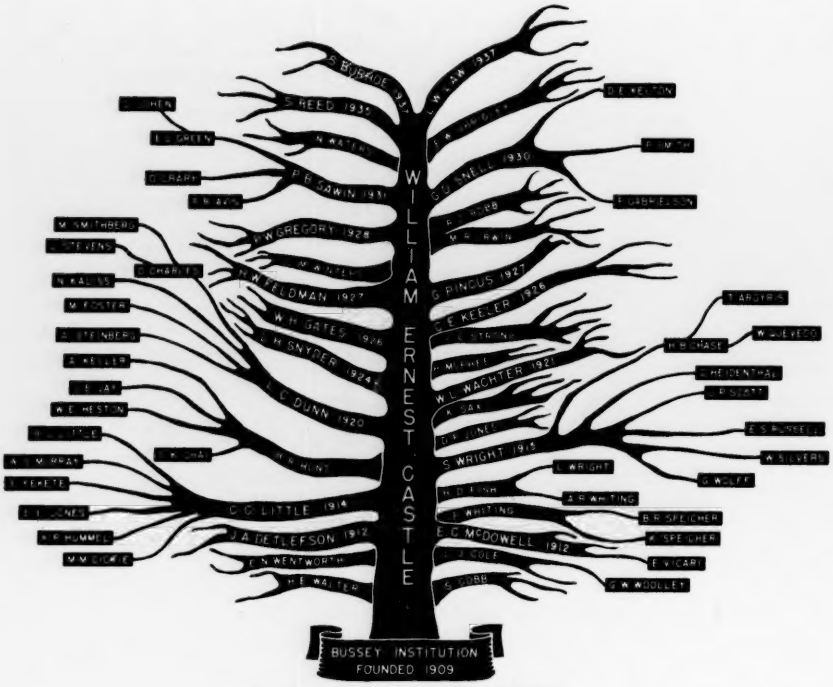
We have been greatly honored by Dr. Castle in this 25th anniversary year of the Roscoe B. Jackson Memorial Laboratory. He flew all the way from California to Maine in June to address our symposium, "25 Years of Progress in Mammalian Genetics and Cancer." Many of the individuals whose scientific development had been most strongly affected by him were among the approximately 225 attendants at this symposium. It seems appropriate at this time to attempt to trace the course of his effect upon them and, as far as is possible in a limited space, upon all of mammalian genetics.

We have attempted to show these relationships on a tree whose stalwart trunk bears the name of William Ernest Castle (Figure 1). All of the good qualities of this tree, the artistic arrangement and excellent draftsmanship, may be attributed to Margaret M. Dickie, research associate of the Jackson Laboratory, and Philip Montella, staff artist. You may blame all possible errors on the author of this article.

Names of the 16 fortunate men who completed work for the Doctor of Science degree with Castle have been placed in large print on the biggest branches of the tree, represented as coming from the front side of the trunk. Following each of these names is the year in which he received his degree. Eight of these men were present for the Jackson Laboratory symposium. Starting with the earliest branches, these are Dr. C. C. Little, founder and director of the Jackson Laboratory; Dr. Sewall Wright, Ernest D. Burton Distinguished Service Professor of the University of Chicago; Dr. Laurence H. Snyder, Professor of Medical Genetics and Dean of the Graduate School of the University of Oklahoma; Dr. William H. Gates, Professor of Zoology, Entomology, and Head of the Department of Louisiana State University; Dr. George D. Snell, Research Associate and past Staff Scientific Director, Jackson Laboratory; Dr. Paul B. Sawin, Research Associate of the Jackson Laboratory; Dr. Sheldon C. Reed, Director of the Dight Institute of the University of Minnesota; and Dr. Lloyd W. Law, Head of Leukemic Studies, National Cancer Institute.

Other scientists worked for a time with Castle at the Bussey, without completing a degree under his tutelage. Many of these feel he contributed very

*Staff Scientific Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.



THE CASTLE "FAMILY TREE"

Figure 1

Castle's influence upon the science of mammalian genetics is illustrated by the spreading tree. Represented on this tree are all of his doctoral students, other students who worked with him at the Bussey, and the students of these students who were present at the 25th anniversary symposium of the Jackson Memorial Laboratory. Please consult the text for the conventions used in this picture.

significantly to their development. This group includes some who worked on material other than the mammals which were Castle's special interest. The names of these Busseyites have been placed in slightly smaller print on branches appearing to arise from the back side of the trunk. Seven of these men also attended these meetings, including H. D. Fish, H. R. Hunt, D. F. Jones, H. McPhee, Karl Sax, Nelson Waters, and Phineas Whiting.

All of the men present at the symposium who actually worked with Castle are shown in Figure 2. They are few in numbers, but their record makes it clear that their scientific impact has been great.

Moreover, Castle's influence certainly is not limited to this group. Many of his students have in their turn attracted other graduate students and have passed on to them some of the wisdom they acquired first at the Bussey. There are many, many such "second generation" students. It would be impossible to place all of their names on the branches of a single tree. However, we can place many of the symposium attendants in their proper position as leaves on the subsidiary branches of the Castle tree. The 36 names so added give some conception of the extending and continuing Castlian influence. Many of the readers of this article, I am sure, will be proud to place themselves and their co-

DR. CASTLE WITH HIS FORMER GRADUATE STUDENTS

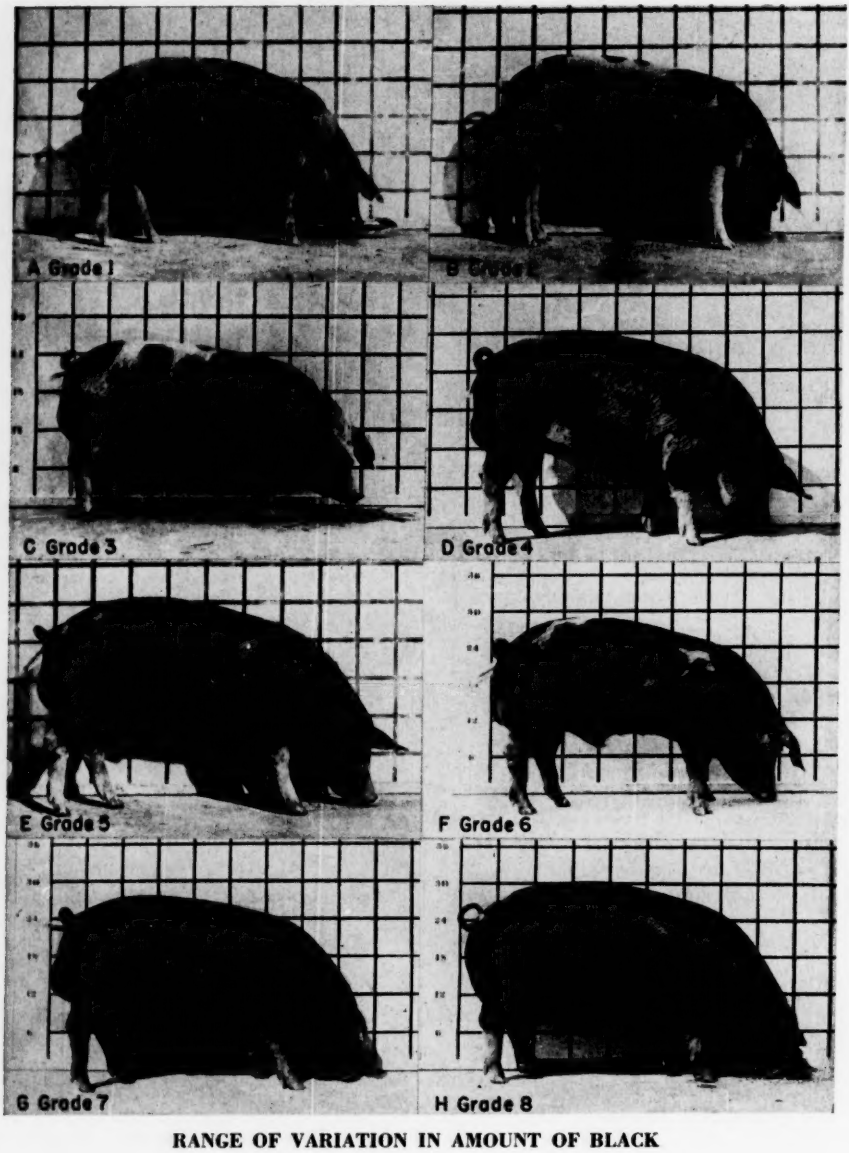
Figure 2

Front row, from left to right: Dr. C. C. Little, Dr. Paul B. Sawin, Dr. D. F. Jones, Dr. Castle, Dr. Hugh McPhee, Dr. Karl Sax, Dr. Phineas Whiting. Back row, left to right: Dr. Sheldon C. Reed, Dr. George D. Snell, Dr. Sewall Wright, Dr. H. D. Fish, Dr. Laurence H. Snyder, Dr. W. H. Gates, Dr. Nelson Waters, Dr. Lloyd W. Law. Dr. H. R. Hunt was not present when the picture was taken.

workers on these same branches.

To the Jackson Laboratory, Castle has made many contributions. It was in the basement of the Bussey, in 1909, that Dr. Little started the inbreeding which led to the oldest of all mouse inbred strains, the DBA. Here, also, "Prexy" carried out pioneer studies on mouse color genetics. The first genetic work on guinea-pigs was a cooperative venture shared by Dr. Castle and Sewall Wright, who even then was developing the original and basic concepts of statistical and physiological genetics which have since inspired so much genetic work. It was at the Bussey that Castle obtained the first evidence of genetic linkage in mammals, between the albino and pink-eye loci in the mouse. Here Dr. George Snell also started his splendid work which has contributed so much to the mouse chromosome map, as well as to many other phases of mouse genetics. Here Dr. Paul Sawin started his work in rabbit genetics. Of our present 32 staff members, 16 clearly owe a part of their scientific development to Castle. Four past staff members present at the symposium also belong on this tree. Because of all this, the corporation of the Jackson Laboratory has elected Dr. Castle as its first Honorary Trustee.

The Jackson Laboratory is proud to pay homage on the occasion of his 87th birthday to William Ernest Castle, a wonderfully sincere, helpful, and encouraging man whose career has provided a firm foundation for the establishment of the whole science of mammalian genetics.



RANGE OF VARIATION IN AMOUNT OF BLACK

Figure 3

Grades of black-spotting in Beltsville No. 1 swine. Pigs of grades 1, 2, 3 and 4 in upper part have less than 50 percent black and those of grades 5, 6, 7 and 8, shown below have more than 50 percent black.

EFFECTIVENESS OF SELECTION FOR EXTENSION OF BLACK-SPOTTING

In Beltsville No. 1 Swine

H. O. HETZER*

THE effectiveness of selection in changing the genetic composition of a population has been extensively explored, and our knowledge of how different genetic situations may influence the progress expected from selection is fairly complete. However, among the many selection experiments that have been conducted with both plants and animals, only a few provide the information needed to test the validity of results expected on theoretical grounds.

Just how rapidly selection can change the mean of a character whose phenotypic variance is influenced by both genetic and environmental factors depends on the heritability of the observed differences, the intensity of the selection and the time interval between generations. With estimates for these three factors as a basis, the effectiveness of the selection practiced can be evaluated by comparing the expected with the actual rate of improvement. Selection for a character such as the one used in the present study can provide valuable information useful in the planning of experiments for the improvement of economically important characters. Even so, the applicability of the methods used to improve a particular character would naturally be limited by the similarity in the genetic basis of variation as well as in the selection intensity and generation interval.

Studies designed to evaluate the effectiveness of selection for quantitative characters in animals have been reported for body size in mice by Goodale,[9] MacArthur,[19] Falconer,[6] Falconer and King,[7] and Lewis and Warwick,[16] for ovarian response to a gonadotrophic hormone in the rat by Kyle and Chapman,[13] for egg production in poultry by Lerner and Hazel[15] and Dempster *et al.*,[3] for fleece and weanling traits in sheep by Terrill[21] and for different economically important traits in swine by Krider *et al.*,[12] Dickerson and Grimes,[5] Dickerson *et al.*,[4] Fine and Winters,[8] and Boulware.[1]

The present paper reports the results of selecting for large amounts of black-spotting in three lines of Beltsville No. 1 swine which were developed from crosses made in 1934 between the Danish Landrace and Poland China breeds. A study of the color genes differentiating the two breeds showed that the black coat pattern of the Poland China behaves as a simple recessive to the white of the Landrace.[11] This study suggested in addition, that the Poland China possesses a number of factors for extension of black, since in the F_2 the pattern breaks up into varying numbers of black spots on a white or sandy ground color. Actually the spotting varied from white or red roan with only two or three black spots to almost black except for a little white on the feet, face and tip of tail. Because of the attention given to individual differences in amounts of black when selecting breeding stock, and in view of the relatively long period of time during which detailed color records were kept on the lines, the data on hand provide a good opportunity for determining how effective selection actually was in increasing the amount of black-spotting. It should be noted that the data used in this study were taken as a routine part of a program, the primary aim of which was to improve such performance traits as fertility, rate and economy of gain and carcass quality.

*U. S. Department of Agriculture, Beltsville, Maryland. The writer is indebted to Mr. J. X. King for recording the color patterns, and to Mr. W. H. Peters for doing the basic computations.

History of Lines and the Breeding Plan

The foundation stocks for the three lines consisted of 15 Danish Landrace sows and five purebred Poland China boars. Four of the Landrace animals were part of a group of 23 head which was imported by the Department from Denmark in 1934, while the remaining 11 animals came from later generations. Most of the Poland Chinas were raised at Beltsville. Except for 23 Landrace animals, 18 boars and 5 sows, which were added during the following eight years, no outside blood was introduced after 1942. By 1951 the herd traced to a total of three Poland China boars, seven Landrace boars and six Landrace sows.

The original breeding plan called for the development of an inbred line similar to the Landrace in all major respects except color. Because of the greater susceptibility of white pigs to sun scalding, the line was to possess the black hair coat of the Poland China instead of the white of the Landrace. However, because of the repeated introductions of Landrace blood following the initial crosses and the fact that the white of the Landrace proved to be dominant over the black of the Poland China, it was necessary to use both white and black-spotted animals until the herd was closed to outside blood in 1942. Beginning in 1942 an increasing proportion of black-spotted pigs were saved for breeding and all litters produced since 1944 came only from matings between black-spotted animals. Also, the amount of black possessed by breeding animals began to increase in 1942, largely because of the increased proportion of black-spotted animals that became available for selection. No entirely black or entirely white animals were ever produced from matings between black-spotted animals.

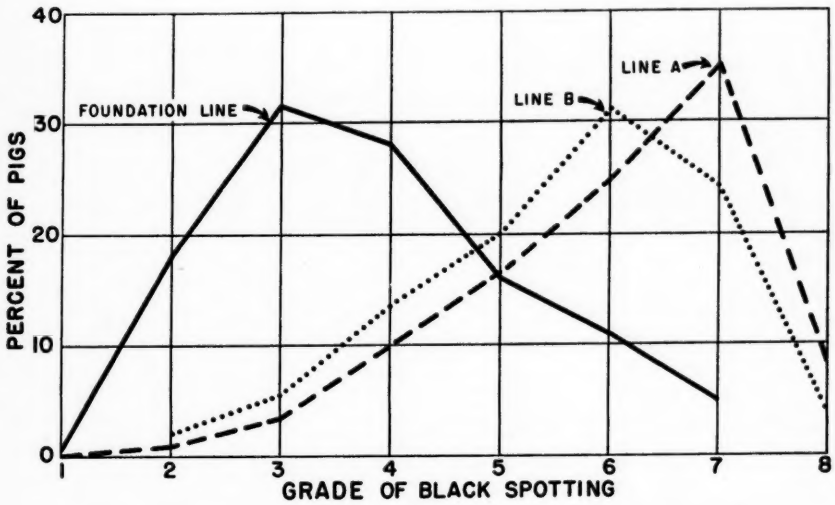
In the fall of 1945 the original line was divided into two non-interbreeding lines, care being taken to maintain sub-lines A and B by the same systems of selection and breeding as those used in the Foundation line. Except for the first five or six generations of the experiment in which the presence of black color constituted the primary criterion of selection, selection in all lines was primarily based on such performance traits as sow productivity, viability, and rate of growth, secondary consideration only being given to individual differences in amount of black-spotting. Thus the selection for large amounts of black was not the maximum possible, but rather that which was thought feasible without materially reducing the selection intensity for the other characters.

Generally from two to four boars were used each season in each of the three lines, each boar usually being mated to a representative group of three or four sows approximately equal to the other groups in age and relationship to their mates. Mating of closely related animals as well as assortative mating based on phenotypic resemblance was deliberately avoided. Thus the inbreeding coefficients should be similar in each case to those expected on the basis of random mating among animals which had no parents or grandparents in common.

All of the pigs produced from matings between black-spotted animals in the 10 years from 1942 through 1951 were included in the analysis. The Foundation line was represented by 757 pigs in 87 litters out of 62 sows and by 20 boars; line A by 1081 pigs in 117 litters out of 83 sows and by 24 boars; and line B by 1024 pigs in 108 litters out of 75 sows and by 26 boars. The pigs from the Foundation line were born between the spring of 1942 and the spring of 1945, while those from lines A and B were born during the same period from the fall of 1945 to the fall of 1951. The number of pigs born in the different years varied from 118 to 225, 63 to 305 and 45 to 326 for the Foundation line and lines A and B, respectively.

The amount of black-spotting was estimated by matching each pig against a series of eight drawings, a grade of 1 corresponding to about five percent black and a grade of 8 to about 95 percent black. With a few exceptions, all grading was done by one and the same person, the grade given each pig being based on a visual estimate of the amount of black on both sides of the body. Grades were obtained at birth and again at weaning. Since the estimates obtained at the two ages were so nearly alike for most pigs, only those at birth were used in the analysis. Photographs illustrating the eight grades are shown in Figure 3.

Figure 4 shows the distribution of the grades of black for the Foundation line and for lines A and B. The three lines varied almost over the entire range, but they differed markedly in mean values. The Foundation line averaged only about 45 percent black, line A at the opposite end had about 70 percent black, while line B had about 65 percent. The distribution curve for the Foundation line is slightly skewed toward the all white region while those for lines A and B lean toward the opposite extreme. This skewness appears to be the greater the more the average amount of black or white deviates from 50 percent, suggesting that a given gene substitution near the extremes of the scale produces smaller visual effects than one near the middle. The situation here is similar to that reported by Wright[22] for the piebald pattern in guinea pigs and by Briquet and Lush[2] for white-spotting in Holstein-Friesian cattle. As heritability estimates or selection differentials based on such data might not be very accurate, adjustment of the grades by means of the angular transformation suggested by Snedecor[20] for percentage data was at first considered. However, since such transformations have little effect when, as in the present study, the primary data are subdivided into as few as eight categories and when few if any of the observations are smaller than five percent or



FREQUENCY DISTRIBUTIONS OF GRADES OF BLACK-SPOTTING

Figure 4

The Foundation line was maintained as a single line until 1945, when it was subdivided into lines A and B. The Foundation line averages about 45 percent black, line A, at the opposite extreme has about 70 percent black, while line B has about 65 percent black.

larger than 95 percent, the grades as originally collected were taken to be sufficiently accurate for the present purpose.

Effect of Sex

Males averaged slightly but significantly less black than females in the Foundation line and line B, but there was no clear evidence of a sex difference in line A. In the combined data, the mean grade of 5.3 for the 1472 males was equivalent to about 59.7 percent black, while the grade of 5.4 for the 1390 females was equivalent to about 61.4 percent. The difference, while small, is statistically significant. The conclusion seems inescapable therefore that the sex difference in amount of spotting is real but too small to be of much, if any, practical importance. Hence no separation was made by sex in the remainder of the study. Lauprecht[14] found sex to have no influence on amount of spotting in black and white piebald cattle. Briquet and Lush[2] in a study of the heritability of amount of white-spotting in Holstein-Friesian cattle, likewise reported no relation to sex. However, Wright[22] found that in guinea pigs males generally had more color than females.

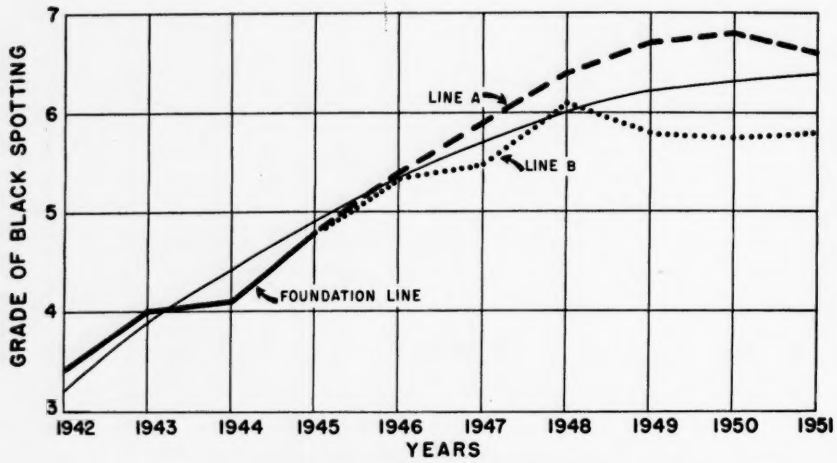
Inbreeding

The average inbreeding of the pigs in the Foundation line rose from 12.0 percent in 1942 to 20.7 percent in 1945. It continued to rise at a fairly uniform rate between 1945 and 1951, at which time the average inbreeding was 37.1 percent for line A pigs and 30.9 percent for line B pigs. These figures correspond to average increases of 2.9, 2.8 and 1.7 percent per year or 3.8, 3.5 and 2.2 percent per generation for the Foundation line and lines A and B, respectively. The average inbreeding of the pigs in the three lines was 18.6, 28.0 and 26.6 percent.

Correlations ranging from 0.13 to —0.08 were obtained between coefficients of inbreeding and black-spotting grades for pigs born in the same year and season. These correlations are quite small and not significant statistically, indicating that variations in amount of black-spotting were not associated with inbreeding. The figures thus suggest that dominance played only a minor, if any, part in determining variations in amount of black-spotting.

Changes in Amount of Black

The annual averages for the black-spotting grades in the Foundation line and in lines A and B from 1942 through 1951 are shown in Figure 5. Starting with an average of 3.4 units of a grade in 1942, the amount of black rose at a fairly uniform rate to 4.8 units in 1945 when the Foundation line was subdivided into lines A and B. Thereafter the amount of black increased to a level of about 6.6 units in line A and 5.8 units in line B. These increases represent a change from about 34 per-



CHANGES IN RESPONSE TO SELECTION

Figure 5

Annual averages for grades of black-spotting in the Foundation line and in lines A and B. The curved line represents the fitted second degree curve for the three lines on a combined basis. The data suggest that the amount of black increased quite rapidly during the early stages of the selection program after which progress became progressively slower.

cent black in 1942 to an average of 70 percent in 1951.

A linear regression fitted to the data indicates an average annual increase in amount of black of .35, .32 and .12 units of a grade in the Foundation line and in lines A and B, respectively. While none of these regressions departs significantly from linearity, a second degree regression fitted to the three lines on a combined basis reveals a small but statistically significant departure from linearity for the entire period. As the curved line in Figure 5 shows, the amount of black appears to have increased at a gradually declining rate until further progress became negligible toward the end of the period. In view of the expected condensation of phenotypic differences near the ends of the scale, as noted above, it is conceivable that some of this decline was due to lines A and B having reached a level of black-spotting where a given genetic change made a considerably smaller visual impression than the same genetic change made in animals possessing about the same amounts of black and white. As an alternative it might be suggested that the apparent decline was due to a gradual decline in genetic variability, a decline in the intensity of the selection practiced during the last years of the experiment or to a decline in both of these factors.

The fact that line B averages less black than line A, when considered with the fact that the second degree term of the regression on time was only on the borderline of significance when line B was excluded from the analysis, favors the latter explanation. Estimates of heritability and of the intensity of selection actually practiced in lines A and B corroborate this suggestion to the extent that both were lower for line B than for line A, as Tables I and II show. Since the progress

TABLE I. Average annual selection differentials for black-spotting grades of sires, dams and both parents

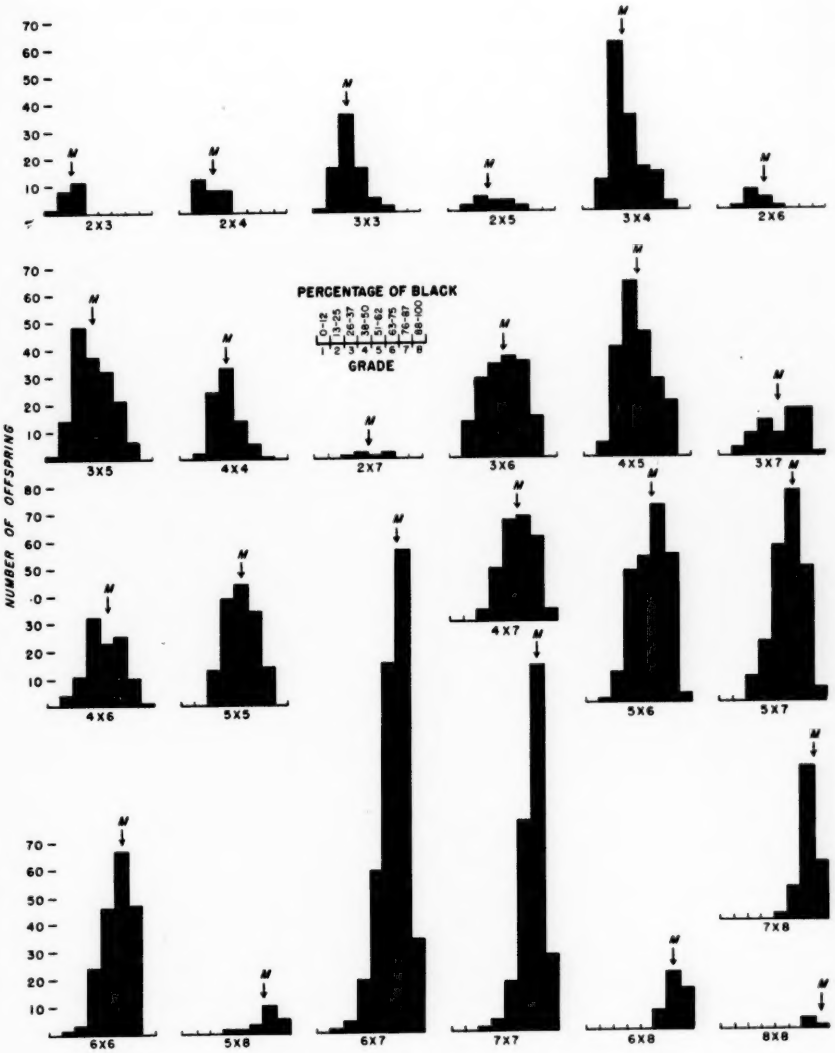
Parent	Line			
	Foundation	A	B	Average*
Sires	0.11	0.72	0.47	0.49
Dams	0.34	0.35	0.32	0.34
Both parents	0.22	0.54	0.40	0.42

*Weighted by number of seasons each line was represented.

TABLE II. Heritability of grades of black-spotting in three lines of Beltsville No. 1 swine

Method of estimation	Line			
	Foundation	A	B	Average*
Paternal half-sib correlation	0.63±.18	0.59±.15	0.52±.14	0.58±.09
Intra-sire regression on dam	0.75±.11	0.83±.09	0.79±.08	0.79±.05
Regression on mid-parent Average*	0.82±.10	0.90±.07	0.64±.06	0.78±.04
	0.76±.07	0.84±.05	0.68±.05	0.76±.03

*Obtained by weighting each of the individual estimates by the reciprocal of its squared standard error.



RESULTS FROM DIFFERENT TYPES OF MATINGS

Figure 6

The distributions of progeny from various types of matings used in the study are illustrated graphically. The grades at the bottom of each distribution represent the different types of matings, a grade of 1 corresponding to 5 percent black and a grade of 8 to 95 percent black. In each case the offspring with little black are at the left while those with much black are at the right. The distributions suggest high heritability of individual differences in amount of black with little evidence of dominance in either direction. Arrows point to mid-parent means.

expected from selection is proportional to the product of heritability and selection intensity, these results warrant the conclusion that the apparent decline in effectiveness of selection was largely due to changes in these factors rather than to a swamping of visual effects towards the end of the scale, as was suggested at first.

Intensity of Selection

The amount of selection practiced for extension of black-spotting was computed in terms of the selection differential or the difference between the individuals used as parents and the average of the group from which they were chosen. The average annual selection differentials for the three lines are shown in Table I. Selection was most intense in line A, followed by line B, and the Foundation line in that order. These differences were due largely to greater line differences in selection differentials for sires than for dams, the average selection differentials varying from 0.11 to 0.72 units for sires but only from 0.32 to 0.35 for dams. The selection differentials over all lines averaged 0.49 for sires and 0.34 for dams, giving a mean selection differential of 0.42 for all parents in the three lines combined.

Young boars and gilts were bred to produce their first offspring at one year of age, while older boars and sows were bred to reproduce at six month intervals following their last litters. The average ages varied irregularly between seasons but were very similar for the three lines, ranging from 1.15 to 1.29 years for sires, from 1.32 to 1.47 years for dams and from 1.29 to 1.31 years for sires and dams. The average generation interval for the three lines combined was 1.21 years for sires, 1.40 years for dams, and 1.30 years for both parents.

Except for the Foundation line in which there was practically no selection for black-spotting among tried sows when choosing those to be used for a second or third litter, the results between lines were remarkably consistent in that selection intensity among mature boars and sows was practically as great, on the whole, as among young boars and gilts. These results are interpreted to mean that relatively greater emphasis was placed on black-spotting when choosing boars and sows for a second or later breeding season, because the proportions retained in culling tried boars and sows were considerably larger than those retained in culling young boars and gilts. Thus approximately 54 percent of all boars and 58 percent of all sows were retained in culling animals that had been used as breeders previously, while only eight percent of all weanling boars and 23 percent of all gilt pigs weaned were saved for breeding. These latter proportions were quite similar for the three lines, varying from seven to nine percent for boars, and from 20 to 26 percent for gilts.

Heritability of Black-Spotting

Estimates of the heritability of individual differences in amounts of black were obtained from (1) the correlation between the grades among paternal half-sibs, (2) the intra-sire regression of offspring's grade on dam's grade and (3) the regression of offspring's grade on mid-parent's grade. Because of the observed time trends in black-spotting and in order to minimize any contributions from environmental effects which varied from season to season, both the correlations and the regressions were computed for each line on a within year-season basis. Therefore, the heritability values obtained represent the fractions of the observed variances which are due to heritable differences between pigs belonging to the same line and born in the same year and season.

Table II presents the several estimates of heritability with their standard errors for each line by each of the three methods. The averages for each line and each method were obtained by weighting each of the individual estimates by the reciprocal of its squared standard error, as suggested by Hazel and Terrill.[10]

Under random mating and in the absence of environmental contributions to the likeness of half-sibs or of parents and offspring, the paternal half-sib correlation would be expected to include one-fourth of the additive genetic variance, plus a small fraction (one-sixteenth or less) of the epistatic variance. It should not be affected by selection except for whatever effect such selection would have in changing the variability of the next generation.[18] The intra-sire regression of offspring on dam contains one-half of the additive genetic variance and something less than one-fourth of the epistatic effects. The regression of offspring on mid-parent includes all of the additive genetic variance plus something less than one-half of the epistatic variance. Thus in non-inbred populations the paternal half-sib correlation and the intra-sire regression of offspring on dam must be multiplied by four and two, respectively, to obtain heritability estimates comparable to those deduced from the regression on mid-parent.

None of the three methods provides any information on the relative importance of dominance, since dominance does not contribute to the likeness of half-sibs or that of parent and

TABLE III. Expected and actual rates of gain in grade of black-spotting

Observation	Line			
	Foundation	A	B	Average
Expected annual gain				
1	0.14	0.32	0.21	0.24
2	0.16	0.45	0.32	0.33
3	0.18	0.49	0.26	0.33
Average	0.17	0.45	0.27	0.32
Actual annual gain	0.35	0.32	0.12	0.35

offspring. However, when applied to single partially inbred lines, all of them underestimate heritability as compared with that found in random bred groups. Since estimates obtained by the above methods would tend to be equally affected by inbreeding, no attempt was made to correct the present estimates for the effects of inbreeding. Also, in so far as deviations from random mating occurred in the development of the three lines, the heritabilities actually obtained would be expected to yield more accurate estimates of expected genetic gains or effectiveness of selection than heritabilities adjusted to a non-inbred basis.

A glance at Table II shows that the various estimates of heritability were quite high, the over-all average being 0.76 with a range in individual estimates from 0.52 to 0.90. Even though the several heritabilities were high for the most part, those deduced from the correlation between paternal half-sibs were lower in all cases than the heritabilities deduced from the intra-sire regression of offspring on dam or those deduced from the regression on mid-parent. However, only in the case of the estimates for the three lines combined was the heritability based on the correlation between paternal half-sibs significantly lower than the corresponding estimates by the other two methods. Since the sires used in the several lines were to some extent selected on the basis of their own degree of spotting, the lower heritabilities obtained by the paternal half-sib method could partly be due to a reduction in genetic variance between selected sires, but the data do not suggest that positively. In any event, such selection is unlikely to be great in effect. Epistasis or a larger environmental contribution to the parent-offspring resemblance as compared with that to the likeness between paternal half-sibs are other possible explanations, but again no reliance can be placed on the relative importance of these factors. While other unknown sources of variation may have introduced a slight bias into the estimates obtained by the different methods, it appears that known environmental effects were adequately discounted by the methods used to obtain the various estimates.

The estimates of heritability were not consistently high or low in any line except for line B in which the estimates were lower in all cases than the corresponding estimates in line A. The difference between the two lines is significant only for the estimates based on the regression of offspring on mid-parent although the average estimates for the two lines likewise differ significantly. Taken at face value these results suggest that the heritability of amount of black has actually changed over the period of selection. Thus, using the Foundation line as a point of reference for any changes in heritability following its subdivision into lines A and B, the indications are of an increase in the heritability of amount of black in line A and of a decrease in line B, although none of the estimates for the Foundation line differs significantly from the corresponding estimates for lines A and B. While a decrease in heritability might readily be accounted for by an increased homozygosity from inbreeding, any increase in heritability would be difficult to explain on this basis unless selection contributed to an increase in genetic variance large enough to offset the effects of inbreeding. Although such an increase might occur on theoretical grounds, the available evidence precludes a definite conclusion concerning any increase in heritability. Because of the similarity in the average inbreeding of lines A and B and in view of the similarity of environmental conditions under which the two lines were kept, it would seem that the lower heritability of amount of black in line B was due to some chance fixation of genes in this line which were preserved by selection, thereby automatically contributing to a more rapid reduction in genetic variance in line B than was true for line A.

Expected Rates of Progress

As indicated above, the rate of progress expected from selection can be obtained by multiplying each selection differential with its corresponding heritability. With estimates for both of these factors available for the three lines, each of the selection differentials shown in the last line of Table I was multiplied with each of its respective heritability estimates in Table II and the expected rates of gain tabulated as shown in Table III. The corresponding observed rates, given for comparison in the last line of Table III, are the linear regressions on time mentioned earlier, with the regression of 0.35 units of a score in the last column being derived from the cumulative records for the 10 years from 1942 through 1951.

The agreement between the observed gains and the several estimates of expected gains is only moderately close for the three lines taken individually, but the estimates for the three lines on a combined basis are strikingly similar. As was to be expected from the variation among the heritability estimates for the three lines, the several estimates of expected gains for lines A and B were not quite as uniform as those for the Foundation line. Also, most of the estimates of expected gains in lines A and B were larger than their respective observed gains, whereas the reverse was true for all three estimates in the Foundation line. If taken at face value, these results suggest that the rate of progress was relatively (as well as absolutely) greater in the Foundation line than in lines A and B, and thus that it was relatively greater in the earlier than in the later generations, since the Foundation line gave way to lines A and B following its subdivision in 1945. Epistatic interactions, if at all important, could account for the apparent discrepancies, for, as pointed out by Lush[17] they would tend to increase the effectiveness of selection at first, but would tend to decrease

it with each generation of segregation. The fact that two of the three estimates of expected gains based on the heritabilities computed from the correlations between paternal half-sibs agreed more closely with their respective observed gains than most of the remaining ones is compatible with this interpretation, because epistatic effects are expected to contribute less to the correlation between paternal half-sibs than to the resemblance between parents and offspring. Since the data afford no means for testing the significance of any of the differences between the expected and the observed gains, the evidence concerning the importance of epistatic effects or the reality of those differences naturally remains inconclusive. Taken over all lines, the close agreement between the expected and observed gains clearly shows that the observed rate of increase in amount of black-spotting could have been predicted quite closely on the basis of the heritabilities and selection differentials obtained from the data.

Results of Different Types of Matings

The distributions of grades among the offspring of the various phenotypically different types of matings are shown in Figure 6. Although the numbers are small for some matings, those between animals classed as possessing the same or nearly the same amount of black generally resulted in offspring clustering about the mean of their parents, while those between animals near opposite extremes resulted in offspring which were almost exactly intermediate in most cases. The results thus are consistent with the suggestion that dominance played only a minor if any part in these data, the indications being that genes acting in an additive fashion are primarily responsible for individual variations in black-spotting. No clear evidence was obtained of the number of genes involved, except that the amount of variability among the offspring of animals in or near the same class would seem to be less than one would expect from the segregation of only one or even two genes. It may be assumed, therefore, that at least three pairs of genes are involved, although there may be only two genes with major effects and many genes with minor effects. Briquet and Lush[2] reported that in their sample of Holstein-Friesian cattle extent of white-spotting is determined by at least four pairs of factors, and that the number of genes with major effects could not exceed 20.

Summary

The effectiveness of selection for large amounts of black-spotting was studied using the records of three lines of Beltsville No. 1 swine from 1942 through 1951. The three lines were descended from crosses made in 1934 between the Danish Landrace and Poland China breeds, with one of the lines constituting the foundation stock from which the other two lines were separated in 1945. Data on 2862 pigs with their parents are included in the analysis.

Females were significantly darker than males, but the difference was too small to need correction.

Inbreeding was found to have no influence on amount of black-spotting, indicating that dominance played only a minor if any part in causing variations in black-spotting.

The amount of black-spotting over all lines increased at a slightly but significantly decreasing rate during the 10 years of selection. This apparent decline in response to selection was due largely to one of the two lines formed in 1945 averaging consistently lower in amount of black than the other line.

Estimates of the heritability of individual differences in amount of black-spotting varied from 0.84 to 0.67 between the three lines, the over-all average being 0.76. Since these estimates were obtained without correcting for the non-randomness of the mating system or for the coarseness of the classification of extent of spotting, it appears that in adjusted data the heritability of amount of black-spotting would be even higher than is indicated by these figures.

Expected rates of progress from selection, computed as the product of each selection differential with its corresponding estimate of heritability, agreed only moderately well with corresponding actual rates obtained for each line, but the estimates for the three lines combined agreed as closely as could reasonably be expected.

The distributions of progenies from various phenotypically different types of matings suggested that two or possibly three genes with major effects were responsible for the variations in black-spotting, although this could not be determined with certainty.

The results indicate that the heritability of variations in amount of black-spotting is high enough that selection based on individual appearance will lead to more rapid changes than selection on a progeny test basis. However, since differences in black-spotting are only of limited interest to market hog producers, emphasis in selection of breeding stock would generally be greater for such performance characters as viability, rate of growth and carcass quality than for amount of black-spotting.

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CYTOGENETIC ANALYSIS OF A SPONTANEOUS PRE-MEIOTIC MUTATION IN MAIZE

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THE paucity of pre-meiotic mutations in maize has prompted a cytogenetic analysis of one which occurred at the *R* locus.

Materials and Methods

The A_1, A_2, *C*, and *R* loci (independent and complementary) are necessary for the formation of anthocyanin in the aleurone of the maize endosperm. The aleurone will be purple or red depending on the presence of *Pr* or *prpr*, respectively.

A plant homozygous dominant for the above loci was fertilized by pollen from a plant homozygous for the *r*, A_1, A_2, and *C* loci. The resulting ear possessed a sector which had endosperms recessive for the *r* locus (Figure 7), indicating that a pre-meiotic mutation had occurred. Thus the embryo should carry the mutant *r* allele as well as one normal *r* allele.

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The mutant kernels, as well as non-mutant kernels from various areas of the ear, were planted. Microsporocytes were collected from the resulting plants and fixed and stored in proprionic alcohol. Aceto-carmine or orcein smears were made and the plants analyzed for meiotic irregularities. At anthesis pollen samples were collected, and the material analyzed for pollen abortion. For genetic tests, individual plants were selfed. Profuse tillering allowed reciprocal crosses to a homozygous recessive *r* tester as well as reciprocal crosses to plants homozygous dominant for all the anthocyanin loci.

Observations

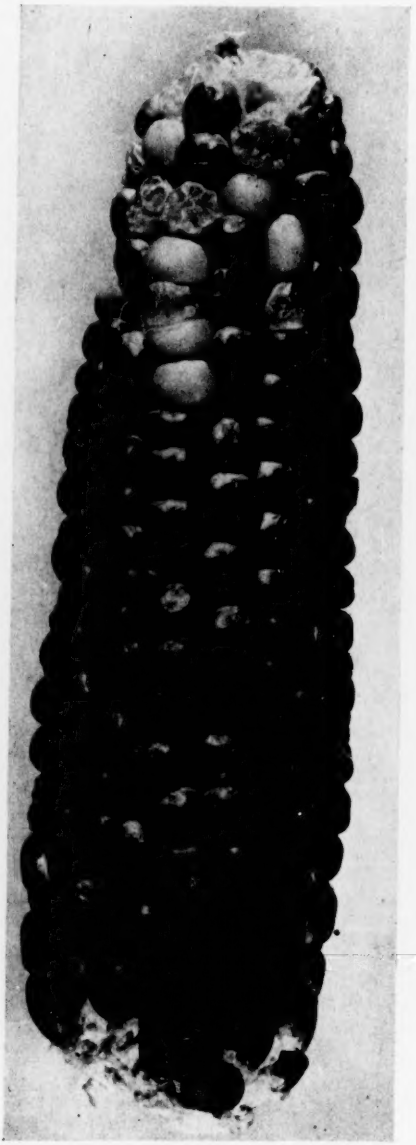
Cytological analysis of meiosis of both mutant and non-mutant plants provided no evidence of chromosomal alteration. Chromosome 10 (on which the *R* locus is located) as well as the other chromosomes appear normal at pachynema. Pollen abortion of both mutant and non-mutant plants was within the range for normal plants. The amount of aborted pollen from individual mutant plants varied from 1.49 percent to 6.48 percent, and from 1.25 percent to 6.99 percent in non-mutant plants.

Selfed mutant plants resulted in a total of 4719 non-anthocyanin endosperms and reciprocal crosses of mutant plants with an *rr* tester resulted in a total of 5832 non-anthocyanin endosperms. No anthocyanin endosperms were found in the above tests, thus mutation of $r \rightarrow R$ did not occur. Reciprocal crosses of mutant plants to a homozygous dominant *R*, A_1, A_2, *C*, *Pr* tester resulted in a total of 3556 anthocyanin endosperms. No $R \rightarrow r$ mutations occurred with either one or two doses of a normal unrelated *R* factor. There was no transmission sterility either through the pollen or egg in the above tests. These tests indicate that no factor, other than the *R* locus itself, was involved and that the mutation is stable.

Non-mutant plants behaved in a normal fashion in genetic tests. Selfs and reciprocal crosses to *r* testers gave ratios expected of normal plants.

Summary

Cytogenetic tests of a spontaneous pre-meiotic $R \rightarrow r$ mutation indicate that it is an autonomous mutation for the *R* locus. No evidence was found for a factor, other than the *R* locus, as a causal agent for the mutation. The mutant behaves as a stable *r* allele. No evidence was found for chromosomal alteration, pollen or transmission sterility. Thus, within the limits of the tests made, it must be called a point mutation.



PRE-MEIOTIC MUTATION IN MAIZE
Figure 7

The A_1, A_2, *C* and *R* loci are necessary for the formation of anthocyanin in the maize endosperm. The ear above possesses a sector which has endosperms recessive for the *r* locus, indicating a pre-meiotic mutation.

AFRICAN VIOLET GENETICS

SHELDON C. REED*

THE African violet has taken the country by storm! And with good reason, for it is an excellent house plant. This sensational invasion of the American home has occurred during the last decade. The *African Violet Magazine* already has over 12,000 subscribers. In view of this extraordinary burst of interest in everything related to the African violet (*St. Paulia*), it is perhaps surprising that only one paper devoted to its genetics has been published.[1]

The present article is an extension of the previous paper. The numbers of plants in the various crosses were not large, as all of the work was done in the writer's home. However, it does not take large populations to show that a characteristic is behaving as a Mendelian dominant or recessive, or that a close linkage between two pairs of alleles is present or absent. It is no longer necessary to present the statistics proving that your F_2 data fit the 3:1 ratio with precision, if such is the case. If the data do not agree with the orthodox interpretations, then they should be published with whatever explanation seems possible. This philosophy will be adhered to in the present paper.

African violets were discovered by the Baron Walter St. Paul in East Africa about 1890. Some of the seeds were sent to Europe and the plants placed on display. Plants were first released to the public in the United States in 1936. All of the dominant mutations, and probably the recessives as well, known in this country, have appeared, *de novo*, since 1936. Several hundred named varieties have been described and many of them are new mutations or combinations of mutations.

The plant has the great advantage for genetic work in that it can be reproduced both sexually and asexually. The widespread commercial and neighborhood practice has been to propagate new plants from the petiole of a rooted leaf. The many named varieties are maintained in this fashion without genetic variability. To obtain new genetic recombinations one must make crosses and grow the plants from the seeds. Such are obtained following hand pollination, as self pollination does not occur in this country except following some accident. The number of plants raised from seeds is small compared with the millions which have been produced from leaf cuttings.

A practical result of the predominantly vegetative reproduction is the discovery of dominant mutations when they occur, while the new recessive mutations can only come to light following sexual reproduction which is relatively infrequent. Consequently it is not surprising that five of the ten mutations studied so far are dominant to the corresponding "wild type" characteristic.

Dominant Genes of African Violets

The mutations studied, which were found to be dominant to the "wild type" are as follows. A symbol has been given for each which is useful for identification purposes.

1. *G* is the symbol for the "girl" mutation. This gene affects the growth habit of the whole plant. It is easily scored at a glance in young plants, due to the continuation of the lack of chlorophyll in the petiole into the base of the leaf with the production of a white spot. The petioles are shorter and more brittle and the leaves are rounder and more scalloped than are the corresponding parts in the wild type plant which is called "boy." Obviously these terms, girl and boy, are not particularly appropriate, as they have nothing to do with the reproductive organs. However, it would be impossible to eradicate the terms so they will have to be endured.

In all of nine crosses of named varieties of the girl type with named varieties of the boy type satisfactory approximations to the 1:1 ratio were obtained. When girl type plants are selfed the offspring are in the proportion of three girl to one boy type. Penetrance is complete, but expressivity is variable and one cannot always distinguish individual girl plants

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DOMINANT "GIRL" MUTATION
Figure 8

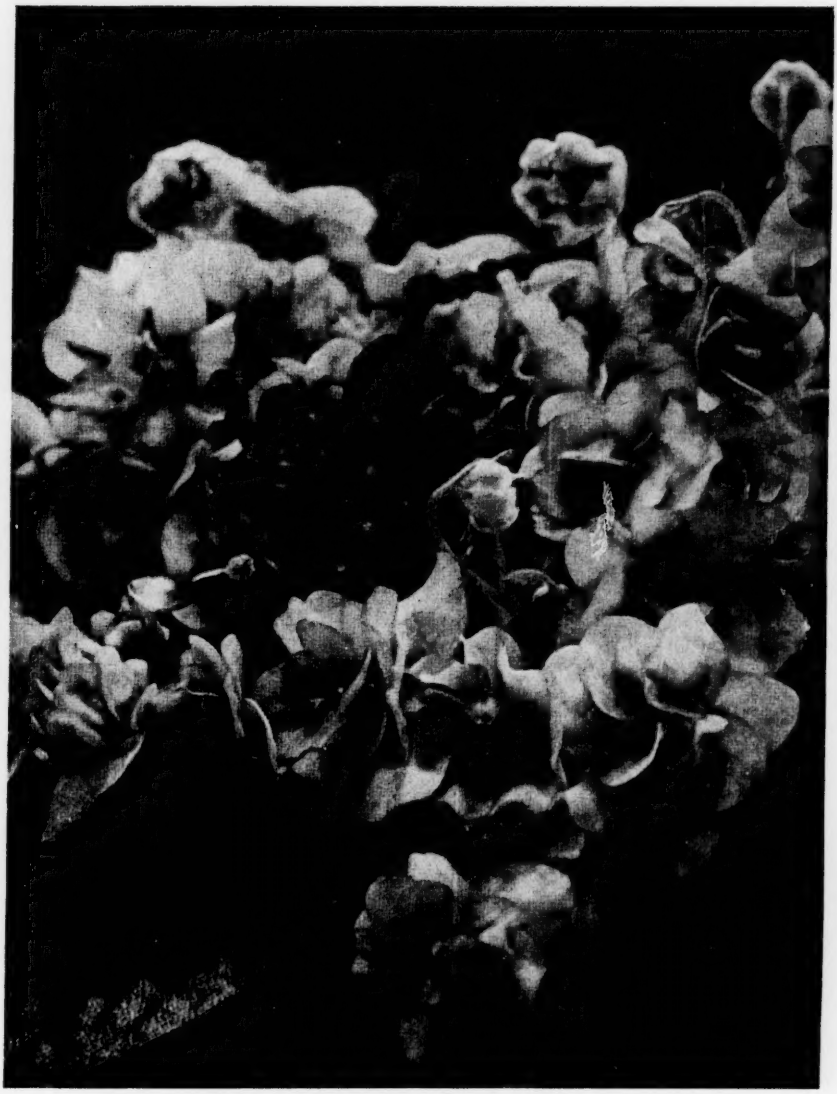
The heterozygous girl type plant (*Gg*). Note the white spot at the base of the leaf blade.

of the genotype *GG* from girl plants of the genotype *Gg* by inspection. The *GG* plants tend to be dwarfed and breed poorly. A plant known to be *Gg* is shown in Figure 8.

2. *D* seems to be a proper symbol for double flowers, the single flowers of the wild type plant being *dd*. Seven crosses of named single varieties with one heterozygous double flowered plant (Double Neptune) gave 58 double to 86 single where 72 of each were expected. The deviation from equality is statistically significant and probably also biologically significant. The double flower characteristic has such extreme variation in its expression as to result in lack of penetrance in some of the plants possessing the gene *D*.

When a plant with double flowers is selfed a reasonable approximation to the 3:1 ratio is obtained though still with a slight deficiency of doubles. Some of the homozygous doubles, *DD*, are probably detectable by inspection, as these plants have many more petals arranged in a more orderly fashion. The flowers of the *DD* plants seem to be much smaller than those of the *Dd* plants. Typical *Dd* flowers are shown in Figure 9.

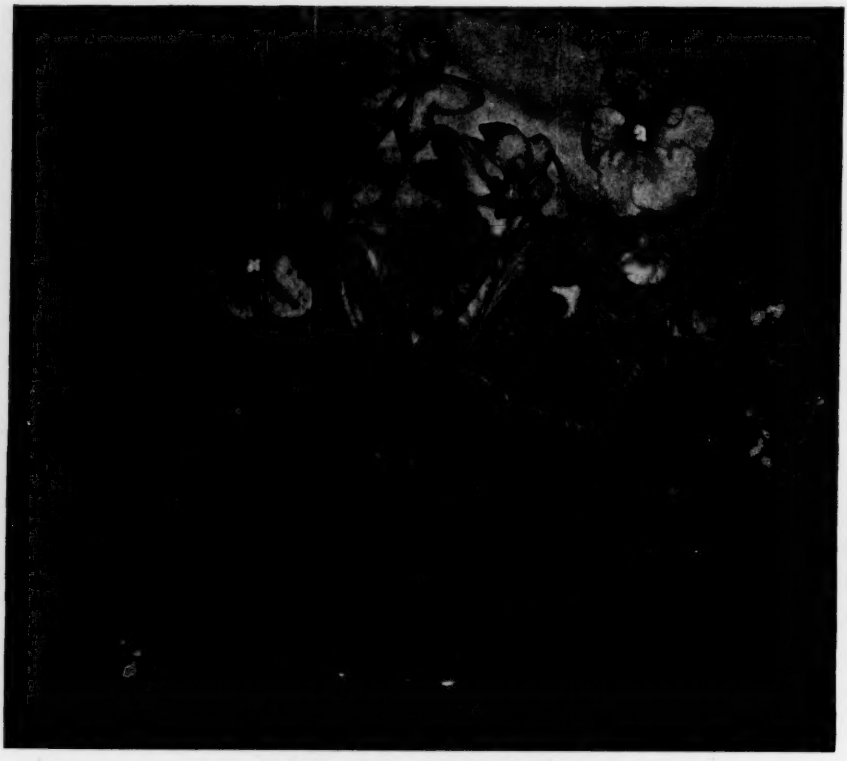
3. *S* is suggested as the symbol for the dominant inhibitor of the pigments, other than



THE DOUBLE FLOWER MUTATION

Figure 9

The heterozygous double flower (*Dd*). This characteristic has extreme variation in its expression which results in a lack of penetrance in some of the plants possessing the *D* gene. *Courtesy of the African Violet Magazine.*



DUPONT RED—A POLYPLOID
Figure 10

The polyploid, Dupont Red, has coarse leaves with prominent hairs. Compare with the smooth leaves shown in Figure 8. *Courtesy of Mrs. Alma Wright.*

chlorophyll, throughout the plant which distinguishes the variety, Snow Girl. The plants can be scored when very small, due to the light green leaves and white petioles. The flowers are usually white though some have areas of blue and still others are completely pigmented, but the pigmentation is never as intense as the wild type purple. The Snow Girl plant used for the outcrosses is heterozygous for the pigment inhibitor. Offspring from the selfing of the plant are not yet in flower so it is not possible to tell whether the still unkown homozygous *SS* plants can be distinguished from the parent *Ss* plant.

4. *A* has been selected to designate the dominant dilution factor which results in the variety, Pink Amethyst. Pink Amythyst is not pink in the sense that Pink Beauty is; it is merely a dilute red. Pink Amethyst is not a favorable characteristic for research as it merely dilutes whatever color would be expressed ordinarily. Scoring colors which vary only in intensity is always hazardous. None-the-less, when crossed with two red varieties it gave one Pink Amethyst (*Aa*) to one red (*aa*). When selfed, it produced a ratio of one red (*aa*) to two Pink Amethyst (*Aa*) to one pale Pink Amethyst (*AA*), the third class being almost white.

5. *M* is used to label still another pigment inhibitor called Mottled. The original plant was a seedling obtained from a hybridizer, Mr. Robert Anderson. It resembles Snow Girl but is not as strong an inhibitor as there is always at least a thin line of color along the top two petals. The anthocyanins of the stems and leaves are inhibited almost completely so the plants can be scored while very young. Some leaves and sometimes all of the flowers "revert" to complete pigmentation. It is not clear what is happening in these unstable plants.

The Mottled characteristic behaves as a clear cut dominant, giving a 1:1 ratio when crossed with unrelated varieties. The plants from the selfing of the heterozygous Mottled parent are not yet in flower, so we don't know whether *MM* is distinguishable from *Mm*.

Recessive Genes of African Violets

The mutations given below were found to be recessive to the "wild type."

6. *r* has been selected as the symbol for what is called red. This recessive, when homozygous, alters the wild type purple to something resembling red, which is perhaps more of a plum color. The penetrance is complete and there is no confusion as to whether a flower is purple or red. Three red varieties crossed with each other, and also selfed, gave 233 plants, all red (*rr*).

The backcross results of 47 non-red to 30 red, equality expected, and the F_2 results of 42 non-red to 11 red show a deficiency of red plants which is not due to faulty scoring but might be due to a differential viability of the two types of plants, or merely to a large sampling error.

7. *sp* is the symbol for spooning. The leaves of the variety known as Gorgeous Bicolor roll up at the edges resulting in a leaf which looks slightly like a spoon. It is only a fair characteristic for genetic research as the spooning is extremely variable and from a selfing, where all the progeny must be genetically homozygous recessives, there were three plants out of 39 which did not show any evidence of spooning. It does not appear at all in F_1 plants, but apears in a little less than one-quarter of the F_2 plants. The modifiers which prevent full penetrance of spooning seem to be both environmental and genetic in nature.

8. The symbol *ge* can be used for the white edging of the flower found in Lady Geneva and the strains derived from it. This characteristic would be interesting for a morphological study, as in some of the plants resulting from a selfing, the edging is expressed as structural damage in the form of notches. All the nicks or notches of a particular plant will be of about the same size. Neither the white edge nor the notches breed true and either may be present without the other, though usually both appear. Among 21 plants from a selfing there were 16 with the edging, notches, or both, while five plants showed neither expression of the character. The Geneva characteristic does not appear in any form in outcrosses to strains without this characteristic, therefore it is recessive in its behavior.

9. The symbol *p* has been adopted for the true pink color seen in Pink Beauty, Pink Cheer and the other shell pink strains. The intensity of the pink may vary considerably on the same plant at different flowering periods. Different segregates from a selfing vary greatly in their "pinkness" which suggests that both genetic and environmental modifiers function. However, in the F_2 from a cross of Pink Beauty Supreme by Double Neptune (purple) there is no difficulty in classifying the progeny in the 3:1 ratio of three purple to one pink. Incidentally, some very nice pink double segregates were obtained from this F_2.

10. The last characteristic will be given the symbol *f*. This stands for Fantasy and represents a strange and interesting gene indeed. Fantasy is an "ever-sporting" strain. The flowers have a basic light lavender color and the cells which mutate give rise to rays and splotches of dark purple. The size of the areas descended from each mutant spot varies from very small to very large on each flower. Each of the flowers on one plant looks different from every other flower but the ratio of mutated area to lavender non-mutated area is about the same for all the flowers on one plant and for the offspring from leaf cuttings of that plant. When a Fantasy is selfed, there is great variation among the offspring, some seedlings showing large areas of purple tissue, others intermediate sized purple areas, others with tiny pinpoint flecks of purple, and still others with entirely purple flowers.

The Behnke Greenhouses very generously sent me a plant of the Fantasy variety before it was generally distributed, so that I have had about four years to work with this characteristic. Even so, the most interesting genetic aspects of this variety still remain to be studied. Some of the current findings follow.

Mutations of the Fantasy lavender gene to a purple allele apparently occur in all tissues of the plant. Florists are well aware that leaf cuttings from a Fantasy plant often produce a plant with only purple flowers. This means that the mutation occurred in the vegetative tissue from which the cutting developed.

In the offspring of the selfing of a Fantasy plant the majority of the plants are Fantasy but the three following types of mutants were also obtained by the writer.

a. Half of the plant was Fantasy and the other half purple. Apparently one of the cells had mutated to purple very early in the development of the embryo, perhaps at the two-cell stage.

b. At fertilization of the egg either the egg cell *or* pollen cell had mutated to purple, but not both. This type of plant has purple flowers due to the fact that the egg *or* pollen nucleus had mutated. However, in these purple flowers there are rays and splotches of a darker purple which show the typical Fantasy pattern. These rays and splotches represent mutations of the second Fantasy gene which had not mutated until the formation of the flowers.

c. When both egg and pollen cell nuclei had mutated by the time of fertilization the resulting plant had all of its flowers with a deep royal purple pigmentation without any pattern of darker areas. All

of the genes at the Fantasy locus are of the mutant stable purple and none is of the old unstable lavender.

When Fantasy is crossed with other strains all the flowers are intense purple without signs of the mutant pattern. Obviously the lavender Fantasy allele is recessive to the wild type purple of other strains. But as the pattern can be seen in plants of type b, described above, it would seem that the purple to which the lavender mutates is not the same as the wild type color but might be said to be "weaker" or lighter purple than the wild type.

In addition, the offspring from the selfing of the Fantasy plant also segregated for a dilution gene which diluted both the lavender and purple areas. Finally, the Fantasy plant carried the ordinary pink gene, *p*, described above.

Polyploidy

Wilson[2] has demonstrated the existence of polyploidy in some commercial strains. Miss Agnes Hansen has made pollen mother cell preparations of my Blue Heiress plant, which show polyploidy of this variety. The writer has crossed the polyploid Blue Heiress with the diploid Double Neptune and other strains. The hybrids clearly have more than the diploid number of chromosomes but much remains to be done with this genetically favorable situation. Cytologically, the African violet is moderately good material but inferior to maize. The haploid chromosome number is 15. Figure 10 shows the coarse nature of the polyploid plant.

Linkage

Crosses designed to detect linkages for all 10 characters described above have been made, but it will be two years before this part of the work will be completed. No linkages have yet been found, but only the double, girl and red genes have been found to be independent of each other, that is, on three different pairs of chromosomes.

Summary

The African violet seems to be a favorable species for genetic research for the following reasons:

1. It is an ideal house plant and vast numbers are grown each year.
2. Reproduction is extremely easy both from cuttings and from seeds. No precautions are needed to prevent self pollination as such does not occur.
3. Hundreds of thousands of plants are produced from cuttings each year. Consequently new dominant mutations are found in large numbers. These will facilitate genetic chromosome mapping.
4. A number of the named varieties are polyploids and crosses with the diploids are successful.
5. The haploid chromosome number is 15. The chromosomes in pollen mother cell division are fairly long but at present do not seem to be as interesting as those of maize.

The modes of inheritance of 10 well known characteristics of the African violet are demonstrated. As this work was done as a hobby, it indicates that very rapid progress with the genetics of African violets could be made if one were to give the work his undivided attention.

Literature Cited

1. Reed, S. C. Abide by Mendel! *African Violet Magazine* 6: No. 4. 1953.

2. Wilson, G. B. A note on the cytology of *St. Paulia*. *African Violet Magazine* 5: No. 2. 1951.

(Received May 4, 1954)

✦

Summer Research Awards for College Faculty Members

The Lalor Foundation announces a new program for 1955 to include 20 summer or interim awards to college and university faculty members for study and research in which chemistry or physics is used to attack problems in any of the biological sciences. Each award will normally not exceed $900 to single men and women and $1,100 to married persons, but is subject to circumstances. The place of work may be at the faculty member's own institution or elsewhere, as may fit the best interests of the program.

It is the hope of the Foundation that not only significant research, but also more dynamic teaching of science may result from this new program and that younger faculty members may find opportunity by this means to advance in their profession.

Inquiries respecting the new faculty summer awards should be directed to C. L. Burdick, Director of the Lalor Foundation, 4400 Lancaster Pike, Wilmington 5, Delaware, by January 15, 1955.

GREGOR MENDEL'S AUTOBIOGRAPHY

WE know that Gregor Mendel as a young man of twenty-eight attempted to pass the state board examination in order to teach biology and physics in a gymnasium (comparable to the American high school) although at that time he had not had any systematic university education. Prepared for this examination only by conscientious but insufficient private studies, it is easy to understand why he failed to pass it. This unhappy experience, however, was beneficial for his future scientific development. One of his examiners, Professor Andreas Baumgartner, physicist at the University of Vienna, recognized the innate ability of Mendel and induced the prelate of the Augustinian convent to send Father Mendel to the Vienna University.

The fact that Mendel attended the University of Vienna (Austria) was first disclosed by Dr. Alois Schindler, Mendel's nephew, in an address delivered on the occasion of the unveiling of the Mendel Memorial at Heinzendorf, Mendel's birthplace.

In his LIFE OF MENDEL (English translation 1932), Hugo Iltis tells us: "But when I applied to the keeper of the University (Vienna) archives to ascertain whether Mendel's name was inscribed in the records for the year 1850-1851, I was informed that his name was not to be found in the roll of students of philosophy whether ordinary or extraordinary. Therefore I travelled to Vienna and scanned the lists with my own eyes, but was unable to find the name for which I was in search. I had almost begun to despair of finding any details regarding Mendel's university career when at length, in the year 1910, Mendel's university certificate of registration at Vienna University turned up in the Altbrünn Augustinian monastery. This document shows beyond question that he was inscribed as an extraordinary student at the philosophical faculty in Vienna for four terms during the years 1851-1853. Through an oversight his name was never entered in the list of extraordinary students. Subsequently, however, I found that he was mentioned in the records of the quæstorship."

Mendel's application for the examination, together with the handwritten autobiography, were apparently found at the same office. The handwritten document which was appended to the request for permission to take the examination, furnishes us with additional authentic and, at the same time, new information regarding the otherwise little-known life story of the young student and priest, Gregor Mendel. The following autobiography was published only in part in Hugo Iltis' LIFE OF MENDEL. Now it is presented here in *its entirety for the first time in the English translation.* Mendel's autobiography in its entirety was published in German for the first time in *Genetica* 8:329-334. The Hague. 1926.

On the following two pages are facsimile reproductions of the second and of the last page of Mendel's Autobiography in his own handwriting.

ANNE ILTIS
(Mrs. Hugo Iltis)

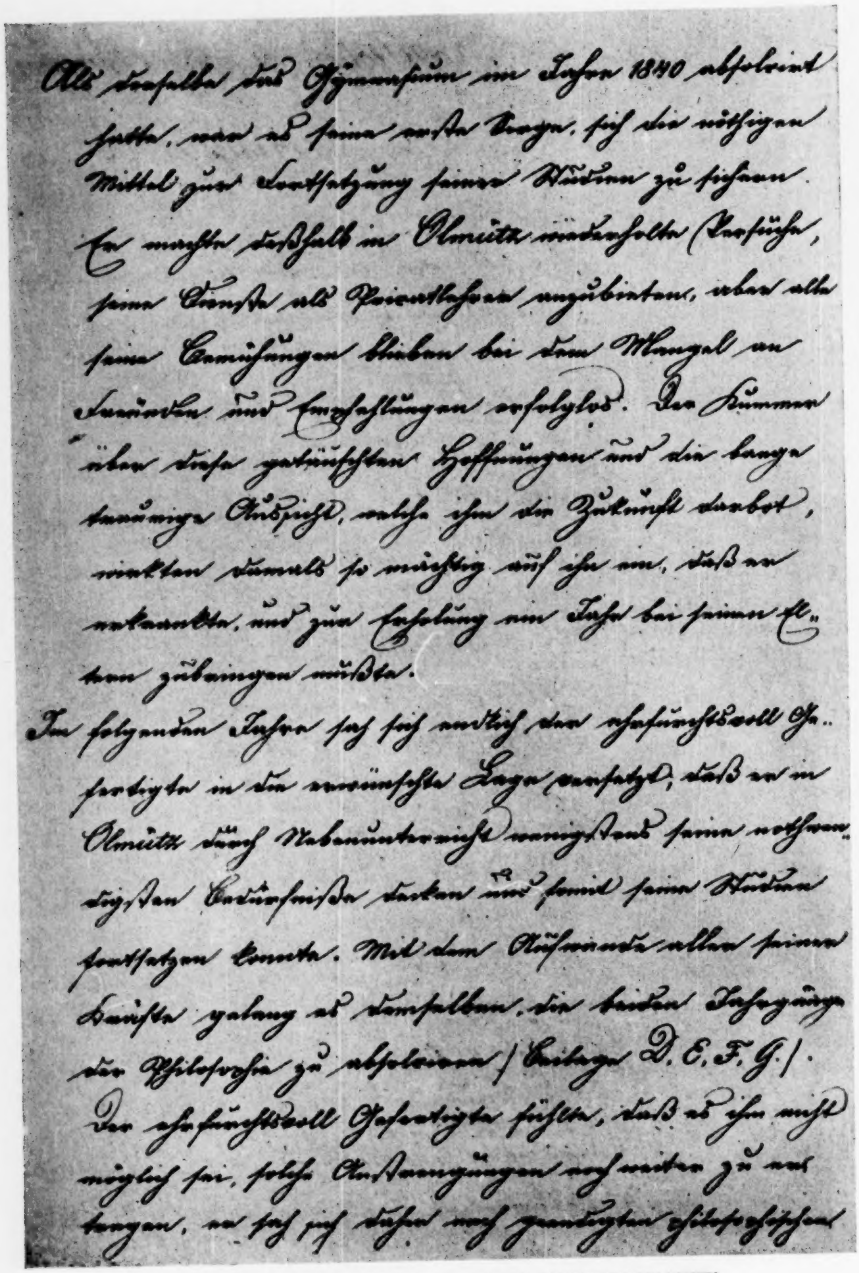
GREGOR MENDEL'S AUTOBIOGRAPHY*

PRAISEWORTHY Imperial and Royal Examination Commission!

In accordance with the high regulations of the Ministry of Public Worship and Education, the respectfully undersigned submits a short sketch of his life.

The same was (in accordance with enclosure A) born in the year 1822 in Heinzendorf in

*Translated from the German by Mrs. Hugo Iltis, Curator of the Mendel Museum, Mary Washington College of the University of Virginia, Fredericksburg, Virginia.

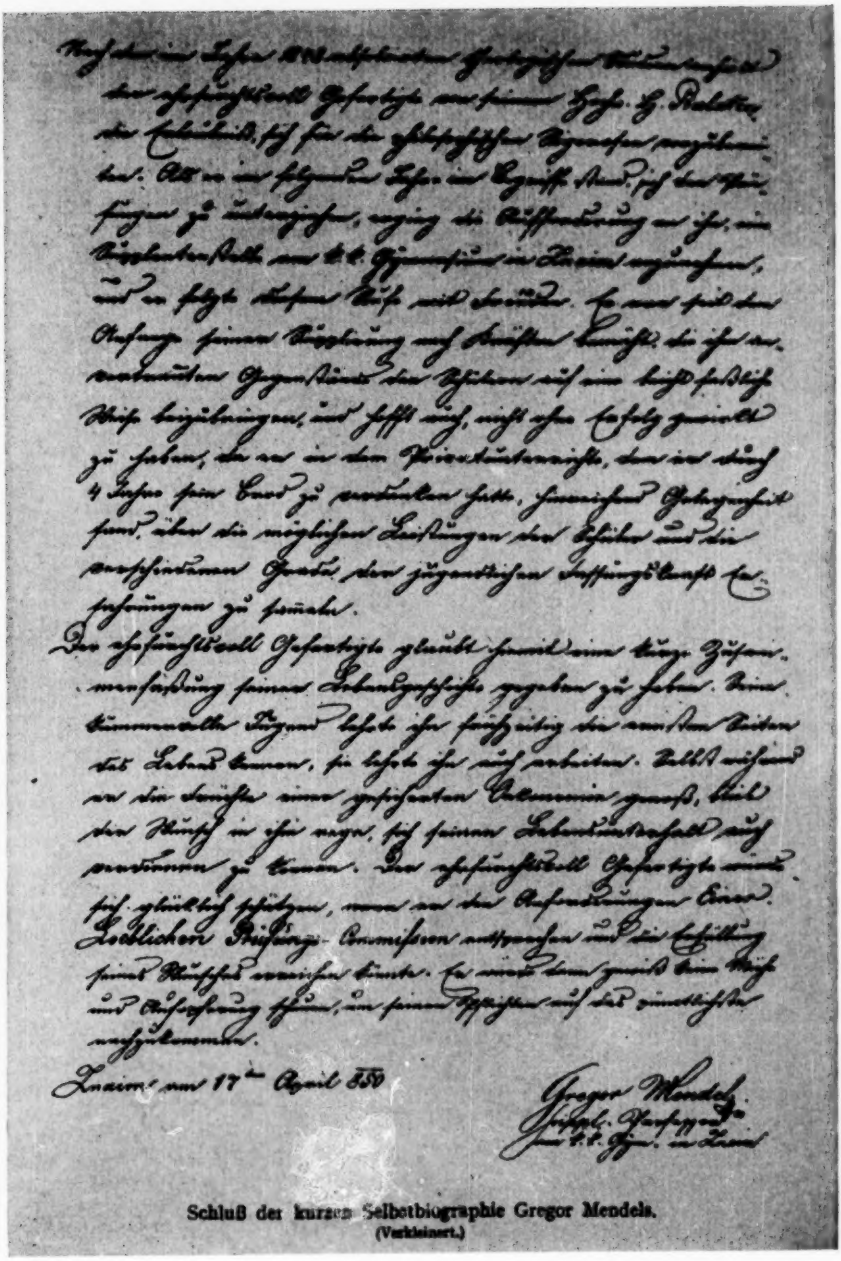


Als daselbst das Gymnasium im Jahre 1840 absolvirt hatte, war es seine erste Sorge, sich die nöthigen Mittel zur Fortsetzung seiner Studien zu sichern. Er musste deßhalb in Olmütz philosophische Vorträge, seine Dienste als Privatlehrer angeboten, aber alle seine Bemühungen blieben bei dem Mangel an Localitäten und Anstellungen erfolglos. Der Kummer über diese getäuschten Hoffnungen und die bange trübsinnige Aussicht, welche ihm die Zukunft darbot, wirkten damals so mächtig auf ihn ein, daß er erkrankte, und zur Erholung ein Jahr bei seinen Eltern zubringen musste.

Im folgenden Jahre sah sich endlich der hochwürdigste Abt bewogen in die erwünschte Lage versetzt, daß er in Olmütz durch Nachunterstützung wenigstens seine nothwendigsten Bedürfnisse decken und somit seine Studien fortsetzen konnte. Mit dem Aufwande aller seiner Kräfte gelang es demselben, die beiden Jahrgänge der Philosophie zu absolviren / Beilage D. E. F. G. /.

Der hochwürdigste Abt erachtete, daß es ihm nicht möglich sei, solche Aufopferungen noch weiter zu ertragen, da sich seine noch jugendlichen Gesichtskräfte

PAGE 2 OF MENDEL'S HOLOGRAPH AUTOBIOGRAPHY



Schluß der kurzen Selbstbiographie Gregor Mendels.
(Verkleinert.)

Silesia, where his father was the owner of a small farm. After he had received elementary instruction at the local village school, and later at the Piarist's College [upper elementary school] in Leipnik, he was admitted in the year 1834 to the first grammatical class of the Imperial Royal Gymnasium in Troppau. Four years later, due to several successive disasters, his parents were completely unable to meet the expenses necessary to continue his studies, and it therefore happened that the respectfully undersigned, then only sixteen years old, was in the sad position of having to provide for himself entirely. For this reason, he attended the course for "School Candidates [applicants] and Private Teachers" at the district Teacher's Seminary in Troppau. Since, following his examination, he was highly recommended in the qualification report (enclosure B), he succeeded by private tutoring during the time of his humanities studies in earning a scanty livelihood.

When he graduated from the Gymnasium in the year 1840, his first care was to secure for himself the necessary means for the continuation of his studies. Because of this, he made repeated attempts in Olmütz, to offer his services as a private teacher, but all his efforts remained unsuccessful because of lack of friends and recommendations. The sorrow over these disappointed hopes and the anxious, sad outlook which the future offered him, affected him so powerfully at that time, that he fell sick and was compelled to spend a year with his parents to recover.

In the following year the respectfully undersigned found himself finally placed in the desired position of being able to satisfy at least his most necessary wants by private teaching in Olmütz, and thus to continue his studies. By a mighty effort, he succeeded in completing the two years of philosophy (enclosures D, E, F, G). The respectfully undersigned realized that it was impossible for him to endure such exertions any further. Therefore, after having finished his philosophical studies, he felt himself compelled to step into a station of life, which would free him from the bitter struggle for existence. His circumstances decided his vocational choice. He requested and received in the year 1843 admission to the Augustinian Monastery St. Thomas in Altbrünn.

Through this step, his material circumstances changed completely. With the comfortableness of his physical existence, so beneficial to any kind of study, the respectfully undersigned regained his courage and strength and he studied the classical subjects prescribed for the year of probation with much liking and devotion. In the spare hours, he occupied himself with the small botanical-mineralogical collection which was placed at his disposal in the monastery. His special liking for the field of natural science deepened the more he had the opportunity to become familiar with it. Despite his lack of any oral guidance in these studies, plus the fact that the auto-didactic method here, as perhaps in no other science, is extremely difficult and leads to the goal only slowly, he became so attached to the study of nature from this time on that he will not spare any effort to fill the gaps that are still present through self instruction and the advice of experienced men. In the year 1846, he also attended courses in agriculture, pomiculture, and wine-growing at the Philosophical Academy in Brünn (enclosure, H, I, K).

After completing the theological studies in 1848, the respectfully undersigned received permission from his prelate to prepare himself for the philosophical rigorosum [examination for the Doctor of Philosophy degree]. In the following year at the time when he was about to undergo his examination, he was asked to accept the position of a substitute teacher at the Imperial Royal Gymnasium in Znaim, and he followed this call with pleasure. From the beginning of his substitute teaching, he made all efforts to present his assigned subjects to the students in an easily comprehensible manner. He hopes his endeavor was not quite without success since, during that private tutoring to which he owed his bread for four years, he found sufficient opportunity to collect experiences regarding the possible accomplishments of the students and the different grades of their mental capacity.

The respectfully undersigned believes to have rendered with this a short summary of his life's history. His sorrowful youth taught him early the serious aspects of life, and taught him also to work. Even while he enjoyed the fruits of a secure economic position, the wish remained alive within him to be permitted to earn his living. The respectfully undersigned would consider himself happy if he could conform with the expectations of the praiseworthy Board of Examiners and gain the fulfillment of his wish. He would certainly then shun no effort and sacrifice to comply with his duties most punctually.

Znaim, on the 17th April 1850

GREGOR MENDEL
Subst. Professor
on the Imp. Roy. Gym. in Znaim

GENE EXPRESSION AS INFLUENCED BY GENETIC BACKGROUND

As Illustrated by Ventral Tubercles on Cervical Vertebrae of Mice

WALLACE MCNUTT*

HUMAN hereditary conditions, many of clinical importance, are often obscure in their mode of transmission from parent to offspring. It is the purpose of this paper to illustrate some of the genetic mechanisms which lead to difficulty in evaluating human pedigrees by the analysis of a parallel situation in the heredity of the mouse. A difficulty often encountered in human genetics lies in differentiation between closely related clinical conditions. This may lead to overlooking the fact that the clinical condition has two or more independent causal agencies; or once this is discovered it requires some care to obtain an accurate differential diagnosis between the two or more similar syndromes each of which varies and overlaps as to the completeness of its manifestation. This labile nature of a syndrome, in most cases, can be explained by one or more of the following activities of the gene: pleiotropic effects, expressivity, and dominant and recessive action.

Genes are usually identified by their most pronounced effect upon the developing individual and, indeed, many genes are described as producing only one effect. Starting with the initial action there probably is a unit effect of the gene. However, it is almost always identified by a character that is only evident at the end of a long series of developments. Usually by this time the chain of events has led to several identifiable characters and the gene producing them is classed as pleiotropic. A study of the manifold effects of the gene will sometimes but not always give insight into its fundamental action. Pleiotropic effects of a gene vary from one individual to another and this obviously contributes to the difficulty of analysis.

A gene may be fully expressed in any one individual or it may be reduced in varying degree—even to complete absence. Expressivity is the name applied for the degree to which a character is shown in a single individual. This is important in varied animal or human inheritance and must be considered in diagnosis. The idea of expressivity can be extended from the individual to a population in which there are variations in the manifestation of a character although the gene for it (capacity for transmission) is always present. This may be given a statistical measure called penetrance. When a character is present in all individuals (of the proper genotype) the penetrance is stated to be complete. If some individuals fail to possess the character although the gene determiner for it is present, the penetrance is stated to be incomplete. This incompleteness adds to the diagnostic difficulties and produces atypical genetic ratios instead of those expected from the regular segregation of Mendelian factors.

Human pedigrees show many examples of a condition which appears to be inherited as a recessive in some families and as a dominant in others. The question arises as to whether the causal gene is different although clinically appearing to be the same, or whether genetic background differences cause the gene to shift between dominance and recessiveness in particular families. These considerations are important to the understanding of human genetic mechanisms and need further experimental study.

The present experiments have been

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designed to test the effect that different genetic backgrounds have in altering the action of the gene. The gene selected for study is the short-ear gene which produces pleiotropic effects, consisting chiefly of alterations of the shapes and processes of some of the bones and cartilages of the entire skeleton.[4] The genetic backgrounds into which the gene is placed are represented by strains of mice each highly homogeneous within itself but each different from all others. The present report deals with the ventral tubercles of the sixth cervical vertebra.

Materials and Methods

A number of foundation strains have been employed by the author and Dr. E. L. Green for preparing special strains for these experiments. The original matings made by Dr. Green employed the Bagg, P and NB strains.[3] Special strains established by the author were started from the P, BALB/c, and C57BL strains. The method of breeding is given below under breeding procedures, where each isogenic strain is considered.

Skeletons of adult mice were prepared for study after the dried flesh was removed from skinned, evicerated carcasses by dermestid beetles. Observations were made with a dissecting microscope and, although measurements and other data were obtained showing variations in form and size of the vertebral processes, the data here presented merely record the presence or absence of the processes. All comparisons of the data are made between short-eared (*sese*) and normal-eared (*Sese*) litter mates of like sex.

Breeding Procedures

Matings of the mice have been made to obtain various strains in which the short-ear gene is placed in different genetic backgrounds in order to observe its action on the development of the ventral tubercles. The short-ear gene was introduced through the P and NB strains and its normal allelomorph was introduced through the Bagg, BALB/c, and C57BL strains. The exact matings from which the derived strains arose are indicated in each isogenic strain listed below.

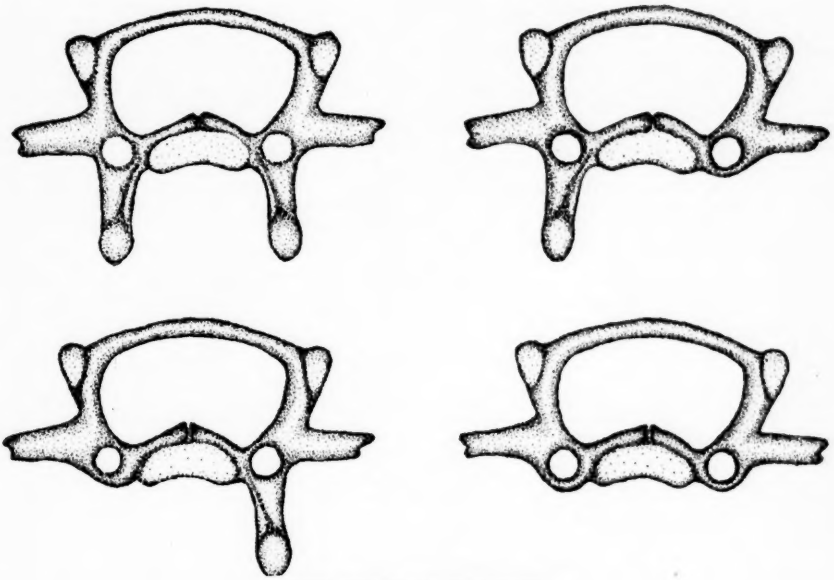
For this type of study it is essential to have animals that are highly homogeneous for all genes except for the gene under study. In mammals this can best be accomplished by inbreeding brother by sister with forced heterozygosis for the gene under consideration, which in this case is the short-ear gene in the mouse [*Sese* (normal-eared) × *sese* (short-eared) littermate]. In *Mus musculus* (20 chromosomes) this type of inbreeding leads to homozygosity for all autosomal genes except that for short-ear and closely linked ones after 20 generations, when, according to the calculations of Bartlett and Haldane,[1] the probability of finding heterozygosis at a locus 1 unit distance from that for short-ear is 0.44. Although mutation will contribute some gene difference, a very close approach to homozygosity is obtained by this method. The possibility of genes closely linked to short-ear remaining heterozygous is further reduced in three of the strains produced. In these strains (SEA-*se*, SEP/1-*se*, SEP/2-*se*) a crossover between the mutant genes short-ear and dilution has occurred and this has been retained in the strain by the selection of the appropriate chromosome. Because these two genes are thought to occupy adjacent loci this eliminates all possibility of heterozygous genes on one side of the short-ear locus.

Listed below are the derived strains from crosses of the foundation strains. The designations of strains are those recommended by the Committee on Standard Nomenclature for Inbred Strains of Mice.[2] Strains SEA-*se*, SEC-*dse* are listed there and strains SEB-*dse*, SEP-*dse*, SEP/1-*se* and SEP/2-*se* are new. Pertinent data are given for each isogenic strain.

SEA-*se* (Syn: Sese-Ab) was originated by Dr. E. L. Green by crossing the Bagg and P strains, both of which were secured from Jackson Laboratory (1946). It was obtained from him in the 20th generation of inbreeding in 1946 and carried beyond the 40th generation at present. Mice in the present data are from the 33rd through the 37th generations. (Gen: *bbdSe/dse*).

SEC-*dse* (Syn: Sese-C) was also originated by and obtained from Dr. Green in 1946 in the 18th generation of inbreeding. This is a strain derived from a cross between the Bagg and NB (maintained at Brown University) strains. For the present study mice were obtained from the 30th through the 34th generations. (Gen: *aabbc*^{a_h} *c*^{a_h}*DSe/dse*).

SEB-*dse* arose from a cross between the P and C57BL strains obtained from Jackson Laboratory in 1946. It is in the 12th generation of inbreeding. The skeletons used are of mice obtained from the 8th through the 10th generations. (Gen: *bbPpDSe/dse*). SEP-*dse* like the preceding strain, received the short-ear gene through the P strain. Both of these strains were obtained from Jackson Laboratory in 1946. This strain has been subdivided into three lines as follows: SEP-*dse* is that portion of the strain in which no crossover between the genes for dilution and short-ear has occurred. It is now in the 20th generation of inbreeding and mice for this experiment were taken from generations 14 through 18. (Gen: *bbppDSe/dse*). SEP/1-*se* was established from a crossover between dilution and short-ear which occurred in the repulsion phase of linkage in the first generation of



SIXTH CERVICAL VERTEBRA

Figure 11

Schematic representation of the sixth cervical vertebra of the mouse (caudal view) showing normal ventral tubercles, and unilateral and bilateral absence of the tubercles.

the above strain. Mice used from this strain were in the 13th through 15th generation. (Gen: *bbppDSe/Dse*). The SEP/2-*se* subline was begun from a crossover between dilution and short-ear which occurred in the coupling phase of linkage in the 10th generation of inbreeding of the SEP-*dse* strain. Mice tabulated here are from the 12th through 14th generations. (Gen: *bbpp dSe/dse*).

Observations

The observations of ventral tubercles on the sixth cervical vertebra were made with particular attention to the effect that the short-ear gene had in each of the different genetic backgrounds. The type of variation noted is indicated schematically in Figure 11. The tubercles are normally large bilateral processes arising from the ventral surface of the vertebra and pointing caudally and ventrally. Immediately dorsal to each is the transverse foramen. The costal element of the vertebra helps to form this foramen laterally. This bar is frequently absent in short-eared mice, converting the foramen into a groove. When this occurs it does not affect the attachment of the tubercle medially, so need not be considered in the present study.

The incidence of this bony process in the isogenic strains of mice is shown in Table I. There are significant alterations in the ventral tubercles of mice depending upon the genetic backgrounds upon which the short-ear gene is acting. In the SEP substrains not a single short-eared mouse had ventral tubercles on the sixth cervical vertebra (number of tubercles possible 134—number present 0). Another observation on this strain is that in normal-eared mice heterozygous for the short-ear gene there is some effect of the gene on the ventral tubercles, in that, in no subline of this strain was the possible number reached (total tubercles possible 56, 54, 20—tubercles present 30, 45, 17 respectively). It should be noted here that the mutant gene dilution does not affect the presence of the tubercles since the incidence is the same in the intense and dilute sublines of this strain.

The effect of the short-ear gene in the SEA-*se* strain appears next in Table I. Here only a few mice homozygous for short-ear had ventral tubercles on the sixth cervical vertebra (total tubercles possible 144—tubercles present 10). Also in this strain mice heterozygous for short-ear did not all possess tubercles (total tubercles possible 122—tubercles present 112).

The next strain to be considered is SEB-*dse*. A full complement of tubercles is not present in either the homozygous or heterozygous mice, but again the short-eared mice are affected to a greater extent (total tubercles possible 46—tubercles present 10) than are the normal-eared siblings (total tubercles possible 44—tubercles present 39).

Mice of strain SEC-*dse* shift further toward the wild population. The short-ear gene does not affect the ventral tubercle nearly as much in this genetic background as it does in the others. Even mice homozygous for the short-ear gene have a high incidence of ventral tubercles (total tubercles possible 108—tubercles present 85) whereas normal-eared littermates that are heterozygous for the short-ear gene never lose the ventral tubercles (total tubercles possible 110—tubercles present 110).

In these strains of mice sex has no significant influence on the development of ventral tubercles. Strain SEA-*se* did show a deviation in that of the 10 short-eared mice having ventral tubercles nine were females and one was a male. This number is too small to give significance to this difference. Nonsignificant deviation occurs in the other direction also as in the heterozygous mice in strain SEP-*dse*. Another method of testing the influence of sex is by combining the observations on mice from all strains which exhibit asymmetrical expression of ventral tubercles (Figure 12). Here again more females show the character than males, but the ratio between the sexes does not deviate significantly in the pooled data.

The asymmetrical expression of the gene seen in the data pooled from all strains is represented in Figure 12. This indicates that more tubercles occur on the left side but the deviation is not significant.

Discussion

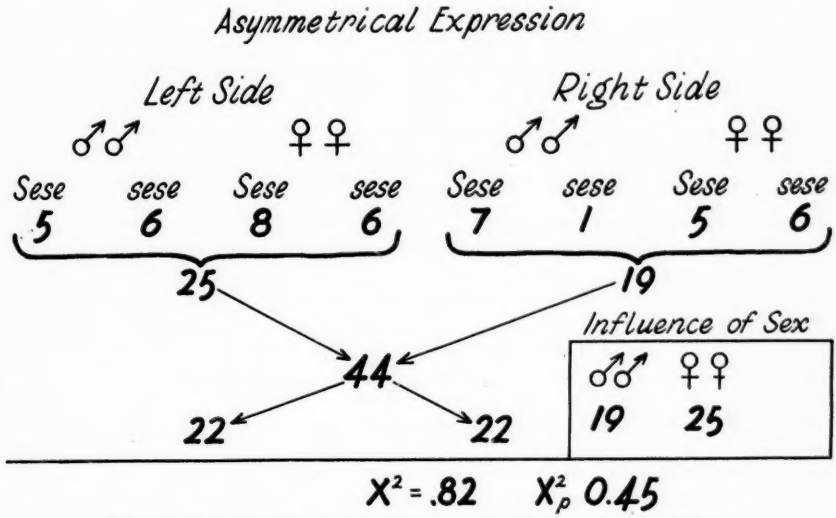
The data that have been presented for the inheritance of ventral tubercles in these strains of mice resemble in three respects the data obtained from the inheritance of many human characteristics. First, typical Mendelian ratios for the presence or absence of this vertebral feature are not obtained; second, dominance and recessiveness varies in the strains; and third, the incidence of the tubercles varies in the different strains.

Using the idea of a threshold in describing these data is illuminating. Figure 13 illustrates the distributions of the homozygous and heterozygous mice in each of the four strains in relation to the threshold for ventral tubercles. Mice below the threshold fail to develop tubercles and those above the threshold do so. Thus homozygous short-eared mice in the SEP substrains all fall below the threshold and have no tubercles, and heterozygous mice of the SEC-*dse* strain all occur above the threshold and all have tubercles. All other groups fall astride the threshold and depending upon the relative numbers in each group below or above the threshold each group possesses fewer or greater numbers of ventral tubercles. Homozygous normal-eared mice are not represented since no mice of this genetic constitution are produced by this mating system (*Sese* × *sese* yields *Sese* and *sese* mice in approximately equal numbers).

It is known that other genetic factors have an effect upon the ventral tubercles. Grüneberg[6] has shown that the gene *undulated* op-

TABLE I. Ventral tubercles on the sixth cervical vertebra

Strain	Genotype	Sex	Number of mice	Number of tubercles possible	Number of tubercles present	Percent present
	dse/dse	♂♂	14	28	0	0.0
		♀♀	14	28	0	0.0
			28	56	0	0.0
SEP-*dse*						
	DSe/Dse	♂♂	12	24	17	70.8
		♀♀	16	32	13	40.6
			28	56	30	53.4
	Dse/Dse	♂♂	13	26	0	0.0
		♀♀	16	32	0	0.0
			29	58	0	0.0
SEP/1-*se*						
	DSe/Dse	♂♂	11	22	17	77.3
		♀♀	16	32	28	87.5
			27	54	45	81.4
	dse/dse	♂♂	4	8	0	0.0
		♀♀	6	12	0	0.0
			10	20	0	0.0
SEP/2-*se*						
	dSe/dse	♂♂	6	12	11	91.6
		♀♀	4	8	6	75.0
			10	20	17	85.0
	dse/dse	♂♂	30	60	1	1.6
		♀♀	42	84	9	10.7
			72	144	10	7.0
SEA-*se*						
	dSe/dse	♂♂	21	42	35	83.3
		♀♀	40	80	77	95.1
			61	122	112	92.6
	dse/dse	♂♂	14	28	5	18.0
		♀♀	9	18	5	28.0
			23	46	10	21.3
SEB-*dse*						
	DSe/dse	♂♂	15	30	26	86.6
		♀♀	7	14	13	93.0
			22	44	39	88.2
	dse/dse	♂♂	30	60	47	78.3
		♀♀	24	48	38	80.0
			54	108	85	78.7
SEC-*dse*						
	DSe/dse	♂♂	30	60	60	100.0
		♀♀	25	50	50	100.0
			55	110	110	100.0



VENTRAL TUBERCLES ON THE SIXTH CERVICAL VERTEBRA

Figure 12

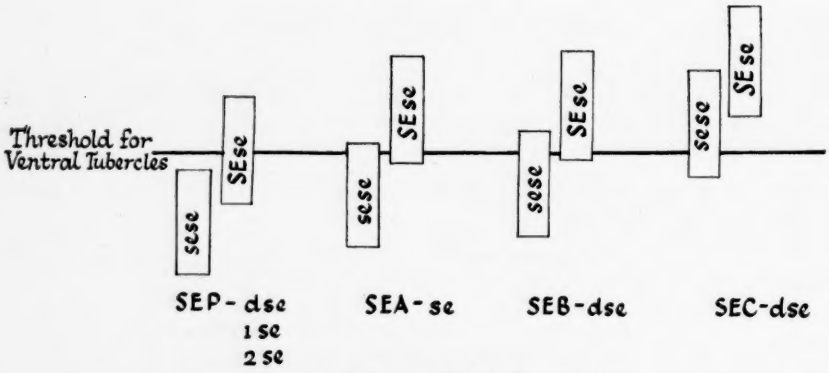
The asymmetrical expression of the gene from all strains indicates that more tubercles occur on the left side, and that more females show the character than males. However, the difference between the sexes does not deviate significantly in the pooled data.

erates to remove this process in some mice. Also the C57BL strain apparently has a genetic background critical for the presence or absence of this feature of the sixth cervical vertebra. Grüneberg[5] noted this in his mice of this strain and it is present in the mice of the same strain obtained from Jackson Laboratory in 1946. Noteworthy is the fact that in the SEB-*dse* strain, which had the C57BL strain as an original parent, the distribution of ventral tubercles is intermediate in the four strains studied. This causes one to think that the processes operating on ventral tubercles in the C57BL strain are different from those operating on the tubercles in short-eared mice. Comparative anatomists also point out that the presence or absence of ventral tubercles is a significant feature in distinguishing the skeletons of some members of the primates. It would appear that here again separate factors are operating.

On the other hand the present experiments show that dilution has no effect upon the expression of ventral tubercles. The SEP substrains illustrate this where two crossovers have occurred between short-ear and dilution, one in the coupling phase and one in the repulsion phase. Thus the three sublines differ in regard to the gene for dilution in that SEP-*dse* is heterozygous for dilution, an autosomal recessive, causing all normal-eared mice to be intense and all short-eared mice to be dilute; whereas SEP/1-*se* is homozygous for intense and thus all mice are intense, and SEP/2-*se* is homozygous for dilution and thus all mice are dilute. Yet in all three of these sublines the expression of ventral tubercles is essentially the same.

It is pertinent to ask whether mutant genes, which might alter the action of the short-ear gene on the ventral tubercles, have accumulated in either of the sublines of the SEA-*se* or SEC-*dse* strains after the division into separate lines in 1946. A tabulation of ventral tubercles in the original SEA-*se* and SEC-*dse* strains by M. C. Green[4] and their occurrence in the sublines reported here indicates that no modifiers of significant magnitude distinguish the separate lines, since the expression of ventral tubercles is similar in each.

Besides reducing the ears, where it acts as a regular autosomal recessive, the short-ear gene produces many other effects, mostly in the skeleton, which do not occur in regular genetic ratios. In a sense the short-ear gene is responsible for producing a syndrome and the absence of ventral tubercles is only one feature of it. This feature of the syndrome is seen to vary in its expression from one strain of mice to another, as a result of the different genetic backgrounds in the different strains. Some features of a syndrome only develop in certain genetic backgrounds as illustrated by the pharyngeal tubercle on the atlas which is



THRESHOLD FOR VENTRAL TUBERCLES

Figure 13

Diagram illustrating the threshold concept for the development of ventral tubercles on the sixth cervical vertebra. Groups falling below the threshold fail to develop tubercles, those above all have tubercles, those on the threshold have varying numbers.

bifid in over 50 percent of short-eared mice in the SEA-*se* strain but perfectly fused in the short-eared mice of the other three strains in this study. The flexibility of the "short-ear syndrome" in mice is shown to depend upon the genetic background. This genetic background is osberved to affect the pleiotropy, expressivity, and dominance and recessive relationship of the short-ear gene.

Conclusions

A genetic study of the incidence of ventral tubercles on the sixth cervical vertebra in four strains of mice, that have been bred with forced heterozygosis for the short-ear gene, illustrate several genetic phenomena.

1. The short-ear gene suppresses the development of ventral tubercles as one of its pleiotropic effects.

2. The amount of suppression varies with the strains of mice, indicating the presence of different modifying factors in the genetic background of each strain. These influence the expression of the short-ear gene modifying the degree of penetrance in each strain.

3. Some of these modifying genes can be thought of as dominance modifiers because the ventral tubercles developed as an irregular dominant character in one strain of mice: in a second strain, under the influence of a different set of modifiers, the character is transmitted as though determined by a recessive gene. Two other strains are intermediate in expression between these.

4. Although other factors than the short-ear gene act on ventral tubercles of the sixth cervical vertebra, the mutant gene dilution has no effect upon its expression in these strains of mice.

5. Absence of ventral tubercles can be presented as one feature of the "short-ear syndrome." As expected this single feature of the syndrome did not occur in a conventional Mendelian ratio, and affected individuals did not possess all features of the syndrome.

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NEW TOMATO SEEDLING CHARACTERS AND THEIR LINKAGE RELATIONSHIPS

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GENES that affect seedling morphology are especially useful tools for cytogenetic investigations. The differences in space requirements of the tomato are such that segregations, which for characters of the mature plant require large areas of land, can be determined accurately for seedling characters in a few flats of soil. The earlier expression of phenotype in seedling characters also permits a considerable saving of time. This great difference in space and time requirements imparts a tremendous advantage to the analysis of linkage groups. New seedling characters are welcome whether they mark new linkages or whether they prove to be linked with previously known seedling markers. In the former instance they expedite further analysis of new groups; in the latter, they can be exploited to advantage to test the effects of various factors on the rates of crossing over. The *Wo-d* linkage in chromosome 2, for example, was found very useful by Soost[9] in his analysis of the effect of asynaptic genes on linkage values.

Another use that has often been proposed for seedling marker genes is to facilitate selection for characters of economic importance with which they might be linked. As an example, recent unpublished work has shown that the useful male-sterile gene, ms_{10} is rather closely linked with *Wo*, thereby permitting selection in the seedling stage for a much higher proportion of male-sterile segregates than would be possible in the normal procedure of propagation by backcrossing.

In view of the rapid rate at which new tomato genes are being discovered, it is not unreasonable to expect that the goal of identifying good seedling markers for each chromosome, and possibly even for each major linkage block, will soon be realized.

Our work has been greatly facilitated by the heritage of a large number of previously discovered mutants. At least 21 have been described, including *a, al, aw, bu, c, d, dl, e, H, hl, l, m, mt, nc, pr, ro, v, w, Wo, Xa,* and *ys*. We present herewith descriptions of four new seedling mutants and results of linkage tests of these and the previously reported *dl*.[7] Each of the four new characters appeared as a spontaneous mutation within lines in which such variations had not been observed previously. None of them is known to be associated with any cytological abnormality.

Description of the Mutants

1. *dv* (dwarf virescent). This mutant appeared spontaneously in line No. 220-17-8 bred by Mr. G. C. Hanna, to whom we are indebted for use of this material. Its outstanding features are reduced growth at all stages of development and pale yellow-green color of the cotyledons (Figure 14) and true leaves of the seedling, changing rather quickly to normal green (Figure 15). The change in color of foliage appears to be most rapid under high levels of temperature, light intensity, and nitrogen supply. The leaf veins and stems of *dv* seedlings often appear more purplish than normal, especially when temperature is low and light intensity high.

Field-grown plants of *dv* are characterized by a slower growth rate, smaller parts and much smaller total plant size than normal. The plants are fairly fruitful under most conditions, although the seed yield tends to be low.

In the presence of *dv* the leaf blade is less finely divided, this effect being more manifest in plants homozygous for *c* (potato leaf). The latter gene also restricts segmentation of the leaf so that the first true leaves of *cc* seedlings are entire, but later leaves develop several segments. If plants are homozygous for both *c* and *dv*, however, the number of entire leaves tends to increase. This interesting interaction is exemplified by the data recorded in an F_2 segregating for *dv* but homozygous for *c* summarized in Table I.

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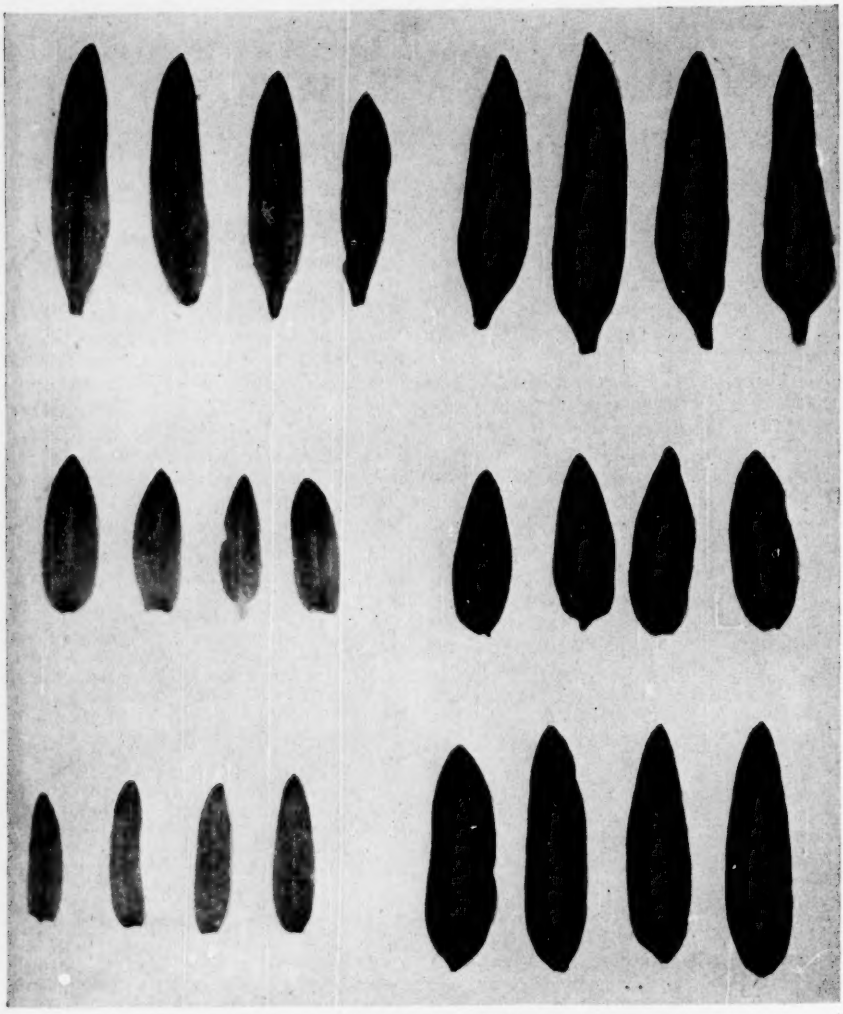


COTYLEDONS OF MUTANT AND NORMAL SEEDLINGS
Figure 14

For each row the four cotyledons in the left group are from mutant seedlings, those in the right group from normal ones of the same segregating population. Top left—reticulate virescent, (*rv*) ; note the darker veins in contrast to all other cotyledons in the figure. Middle left—yellow virescent, (*yv*) ; note the more intense pigmentation at the tips of the cotyledons. Lower left—dwarf virescent, (*dv*).

TABLE I. Effect of *dv* and *c* on leaf development

	(number of plants) Number of entire leaves				
Phenotype	0	1	2	3	4
c dv+	32	82	23	0	0
c dv	1	17	26	3	1

2. *rv* (reticulate virescent). This second virescent mutant was also obtained from Mr. G. C. Hanna, who first observed it in line No. 45-116-4. Under greenhouse conditions it is very easily distinguished from normal; fur-

thermore, it also differs from the other two virescents described here. Its cotyledons are pale green instead of yellow-green in color, and the veins of cotyledons (Figure 14) and true leaves (Figure 15) are markedly darker than the rest of the blade, imparting a peculiar reticulate appearance. The cotyledons are smaller than normal (Figure 14) and the growth of seedlings is very greatly retarded. A ragged leaf margin is associated with these effects on color and shape of leaves. In contrast to these features of plants grown in the winter greenhouse, the same plants under conditions in the field in midsummer grow at an almost normal rate and show very little leaf modification. A high degree of pollen sterility greatly reduces fruit setting under all tested conditions, but *rv* plants function satisfactorily as pistillate parents.

3. *yv* (yellow virescent). This virescent appeared as a second mutant in an asynaptic line, No. 2-94, of the variety San Marzano. Its outstanding characteristic is a bright yellow-green hue that appears in the cotyledons (Figure 14) and in younger leaves regardless of age or environment. Another diagnostic feature is the tendency of the same organs to develop a darker coloration at their extremities (Figures 14 and 15). The transition from this initial pale color to a light green (Figure 15) in the greenhouse and to a normal dark green in the field takes place rather rapidly. Despite weak growth in the greenhouse, *yv* plants can be induced to produce fruits and seeds. Field-grown plants are fertile and normal in all other respects save the yellowing at the growing points.

4. *tf* (trifoliate). This mutant was obtained in 1948 as inbred No. 5701-9-3-4 from Dr. Oved Shifriss, then with the W. Atlee Burpee Co. of Philadelphia, Pa.

This gene affects shape and size of all true leaves apparently with little if any environmental interaction. The first true leaves (Figure 16) appear much narrower and more entire than normal and differ from normal in a characteristic rough texture. Leaves above the fifth position (Figure 17) are usually split into three narrow lobes at the end of a very long petiole, and a few additional lobes occasionally appear. Internodes are short, branches are few, and the plant has a stiff upright appearance. Pedicels and peduncles are somewhat shorter than normal. The plant is generally unfruitful and refractory for hybridization in the field, but it can be readily hybridized in the greenhouse.

Another mutant of similar phenotype, but independent origin was described by Dennett *et al.*[3] The trifoliate phenotype of all progeny of reciprocal crosses between the two mutants establishes beyond any doubt that they are allelic.

5. *dl* (dialytic). This mutant, which has been described previously,[7] can be identified in the seedling stage by the partial suppression and malformation of the large trichromes.

Inheritance

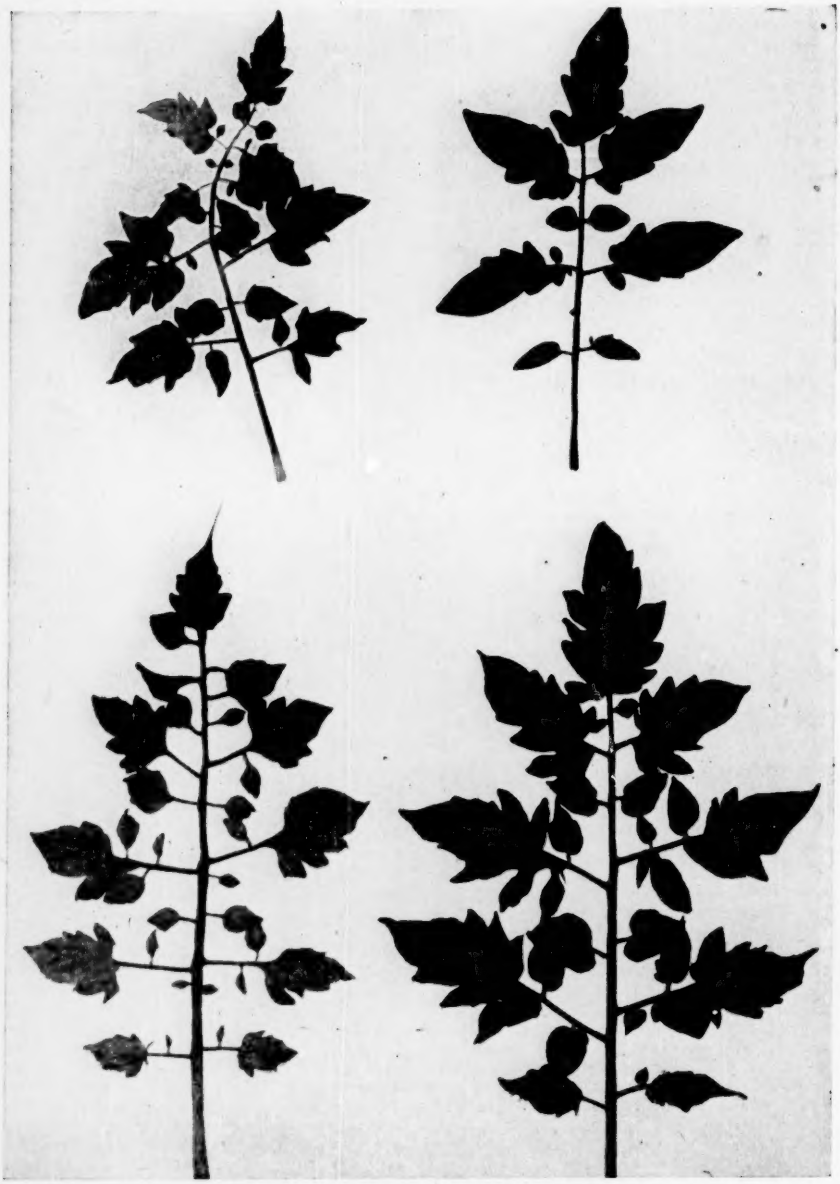
Since the progeny of each mutant phenotype consisted exclusively of mutants, they were homozygous and very likely determined by recessive genes.

Crosses were made between the mutants and many different stocks of the normal type. The progenies, including many plants in the offspring of each mutant, were composed entirely of normal plants.

These indications of the recessive nature of each mutant were also supported by F_2 segregations (Table II),* in which the mean proportion of mutant individuals was 19.1 percent for *dv*, 15.9 percent for *rv*, 21.8 percent for *yv*, 23.8 percent for *tf*, and 20.9 percent for *ll*. In the case of *tf* the total segregation does not deviate significantly from the expected 25 percent, but for each of the other mutants the deficiency is highly significant statistically. Despite these discrepancies it is very probable that each is conditioned by a single recessive gene. The deviations presumably reflect the lower viability known to characterize the homozygous mutants. The rather large heterogeneity χ^2 obtained for all mutants except *dl* and *rv* suggests that at least a part of this deficiency might be incurred in the process of germination. Furthermore, the linkage relations of some of these mutants, presented in the following section, could be explained only if a single gene determined the difference between the normal and mutant conditions.

Variable germination is not likely to be responsible for the unusually low proportion of *rv* because each family deviates from expectancy in approximately the same proportion (Table I), and attempts to alter the proportion by varying percentage germination under different environmental conditions were unsuccessful. The consistent appearance of *rv* segregates in about 16 percent of all F_2 and F_3 progenies hints that the departure from 25 percent might be caused by something that acts more precisely than reduced ability to germinate. Although lowered gametophytic

*Because of space limitations it has been necessary that we omit from the text Table II—(Segregations of Mutant Genes, Tests for Goodness of Fit and Heterogeneity) and Table III—(Tests for Linkage Between Seedling Mutants and Various Genes). These tables are available as supplementary documents. To obtain this material, order Document No. 4406 from the American Documentation Institute, Auxiliary Publications Project, Photoduplication Service, Library of Congress, Washington 25, D. C. Remit $1.25 for microfilm (images one inch on standard 33mm. motion picture film), or $1.25 for photoprints (6 × 8 inches, readable without optical aid).



VIRESCENT MUTANT AND NORMAL LEAVES

Figure 15

Representative mature leaves of the virescent mutants and normal type taken from plants grown in the greenhouse. Upper left—*rv;* note the reticulation effected by darker pigmentation of the veins and paler color of the intervein areas. Upper right—*dv;* note the normal color but reduced size. Lower left—*yv;* note the general chlorophyll deficiency but nearly normal size. Lower right—normal.

TRIFOLIATE MUTANT AND NORMAL
Figure 16

At the left are shown trifoliate (*tf*) and at the right normal seedlings. The first leaves appear much narrower and more entire than normal and have a characteristic rough texture.

viability might contribute to the deficiency, critical test crosses must be made before the frequencies of *rv* can be properly explained.

Improper classification does not likely explain any of the departures from expectation. The phenotypes of all mutants are well distinguished from normal except possibly older seedlings of *dv*, which attain a normal green color save an abnormal yellowing at the growing point. Even with *dv*, however, classifications made in the field, where the identity of this mutant cannot be mistaken, showed the same disturbed segregations.

Single exceptional families do not account solely for the heterogeneity in cases of a high level of significance, for in no case is the heterogeneity χ^2 reduced to a non-significant level by withholding the data of any single family.

Linkage

Exploratory tests for linkage were made by scoring recombination in repulsion F_2 progenies derived from crosses of the five mutants and 11 standard marker genes, presumably belonging to independent linkage groups, and from crosses of the mutants among themselves. In addition, a few other marker genes were tested against one or several of the mutants. Total segregations for each combination are given in Table III, and the results of all tests are summarized diagrammatically in Figure 18.

On applying Mather's linkage detection formula,[6] a significant indication of linkage was found for the following gene combinations: *dv-d* ($\chi^2 = 284.11$), *dv-m* ($\chi^2 = 220.03$), *yv-d* ($\chi^2 = 8.47$), *dl-l* ($\chi^2 = 15.41$), and *dl-bu* ($\chi^2 = 50.41$).

In addition, *tf* was found to be linked with a new wilty gene (which was present in the original stock and is not allelic with the well-known *wt*) with a linkage χ^2 of 213.89.

The departure from independent recombination in all of the above cases was significant in all families contributing to the totals except for *yv-d*, in which only part of the families deviated in the direction of repulsion linkage. This indication of linkage, furthermore, contradicts trisomic tests (unpublished data) that show no relation between *yv* and chromosome 2, which has proved to bear *d* and other genes of linkage group I.[5,8] This apparent disagreement is most likely explained by the fact that classification of *yv-d* individuals is difficult in

TRIFOLIATE PLANTS
Figure 17

Branches from mature plants of *tf* (left) and normal (right). Internodes are short, branches are sparse and the plant has a stiff upright appearance.

the seedling stage. In this connection it is significant that no indications of linkage were encountered in the field, where phenotypes of the mature plants are not confused.

The lack of double recessives in progenies of *rv* and *e* suggests a linkage between these two genes, although the χ^2 (3.200) is significant only at the 10 percent level. According to the standard error of the crossover value calculated by Immer's methods,[4] however, the indication of linkage is highly significant.

Linkage intensities were measured by means of Immer's[4] product method. According to the calculations, the data indicate the following orientation of *dv* on chromosome 2: *dv*-6-*m*-1-*d*, about nine units separating *dv* and *d*. Since *d* and *m* were in coupling phase and both were in repulsion with *dv* in the parent stocks, the estimate for the *d*-*m* interval is more reliable than that of the others. The fact that *dv m d*+ segregates, part of which would represent double crossovers according to this order, were not encountered whereas all other recombination classes were obtained in an F_2 of 1654 individuals suggests that the above order is correct.

Sufficient information was obtained concerning *dl* to define its locus with reasonable certainty. In the first place, a crossover value of 38 ± 2 percent was obtained between *dl* and *l*. Previous work on this chromosome summarized by Butler[2] indicated 27 units between *l* and *bu*. If *bu* were between *l* and *dl* a linkage of about 11 units would be expected between *dl* and *bu*. The latter value was closely approximated by our tests, the observed value being 9 ± 4 units. According to these findings, the order of these three genes is *l*-27-*bu*-9-*dl*. In addition, the gene *al*, which was demonstrated by the trisomic method to exist on this same chromosome,[8] has given us a suggestion, which is not statistically significant, of linkage with *dl*. In previously reported tests *al* was not found to be linked with any other genes. If the *dl*-*al* linkage is real, it would mean that *al is* located some 40 ± 3 units from *dl* opposite *l* and *bu*.

The crossover value for *rv*-*e* is calculated to be 24 ± 2 units.

No linkages were encountered between either *yv* or *tf* and previously described genes. As already outlined, the suggestion of linkage between *yv* and *d* proved to be spurious. The linkage of *tf* and the new gene for wiltiness was characterized by a recombination value of 13 ± 2 percent.

Discussion

In studying the seedling mutants in the progenies reported here and in other studies, we have been impressed by certain of their assets and limitations. Considering the large number of known tomato mutants (108 reported in the latest Report of the Tomato Genetics Cooperative) and the rate at which they are being discovered, discrimination in

Tester gene	*dl*	*tf*	*rv*	*Wo*	*m*	*d*	*r*	*y*	*c*	*a*	*lf-j*	*l*	*bu*	*H*	*Xa*	*ol*	*wt*	*e*
Linkage group				I	I	I	II	III	IV	V	V	VI	VI	VII	VII	VIII	X	XI
Chromosome				2	2	2		1				8	8	10	10	8	7	
dl	–	0	0	0		0	0	0	0	0		***	***	0	0	0		
tf	0	–	0			0	0	0	0	0	0	0	0	0		0		
rv	0	0	–	0		0	0	0	0	0	0	0		0	0	0		?
yv	0	0	0	0		0	0	0	0	0	0	0		0		0		
dv	0	0		***	***	***	0	0	0	0		0	0	0	0	0		

LINKAGE TESTS

Figure 18

Summary of tests for linkage among the seedling genes and between them and previously known chromosome makers. The symbol 0 indicates no significant indication of linkage; ? indicates an uncertain relationship; *** indicates linkage significant at the .001 level. Chromosome numbers are those applied by Barton.[1]

favor of the most useful seedling markers is justified. In our estimation the qualifications of good seedling markers are: (1) ease of identification while in cotyledons or first true leaves; (2) reasonable viability in the seedling stage; and (3) sufficient viability and fertility as mature plants to permit controlled matings and the production of hybrid seed. Generally, however, requirement 1 is in developmental conflict with nos. 2 and 3, for the more drastic the difference in the seedling stage, the greater the tendency toward inviability and/or sterility at maturity. Thus many such sharply distinguished mutants as albinos and certain other chlorophyll deficiencies have not been analyzed because they die in the cotyledon stage or shortly thereafter. Others—*w* (wiry), for example—suffer such deformities of the flower that controlled crosses with them are difficult or impossible. Fortunately, however, the correlation is not absolute, and means can often be devised to obtain controlled crosses with mutants that tend to be weak or partially sterile. Furthermore, it might be anticipated that with additional study, methods may be found to identify in the seedling stage certain mutants that are known now only by their features in later stages of growth.

Summary

Four new tomato seedling mutants, each conditioned by a recessive gene, are described. Three of these—*dv, rv,* and *yv*—are virescent in the respect that they cause a partial breakdown of chlorophyll formation, which is most acute at the growing point and least severe in older leaves. The fourth mutant, *tf*, is characterized by leaves that are reduced in breadth and number of segments. Normal F_2 ratios were obtained for *tf*; a deficiency of homozygous recessives was observed for the others. In all other respects inheritance was normal and segregations well-defined.

Linkage relations were analyzed among the four new mutants and the previously described *dl* and between them and a group of standard linkage markers. No indication of linkage was found for *yv*. The other genes were found to be linked as follows: *dv*-6-*m*-1-*d*; *l*-27-*bu*-9-*dl*; *rv*-24-*e*; *tf*-13—new wilty gene found in parent stock.

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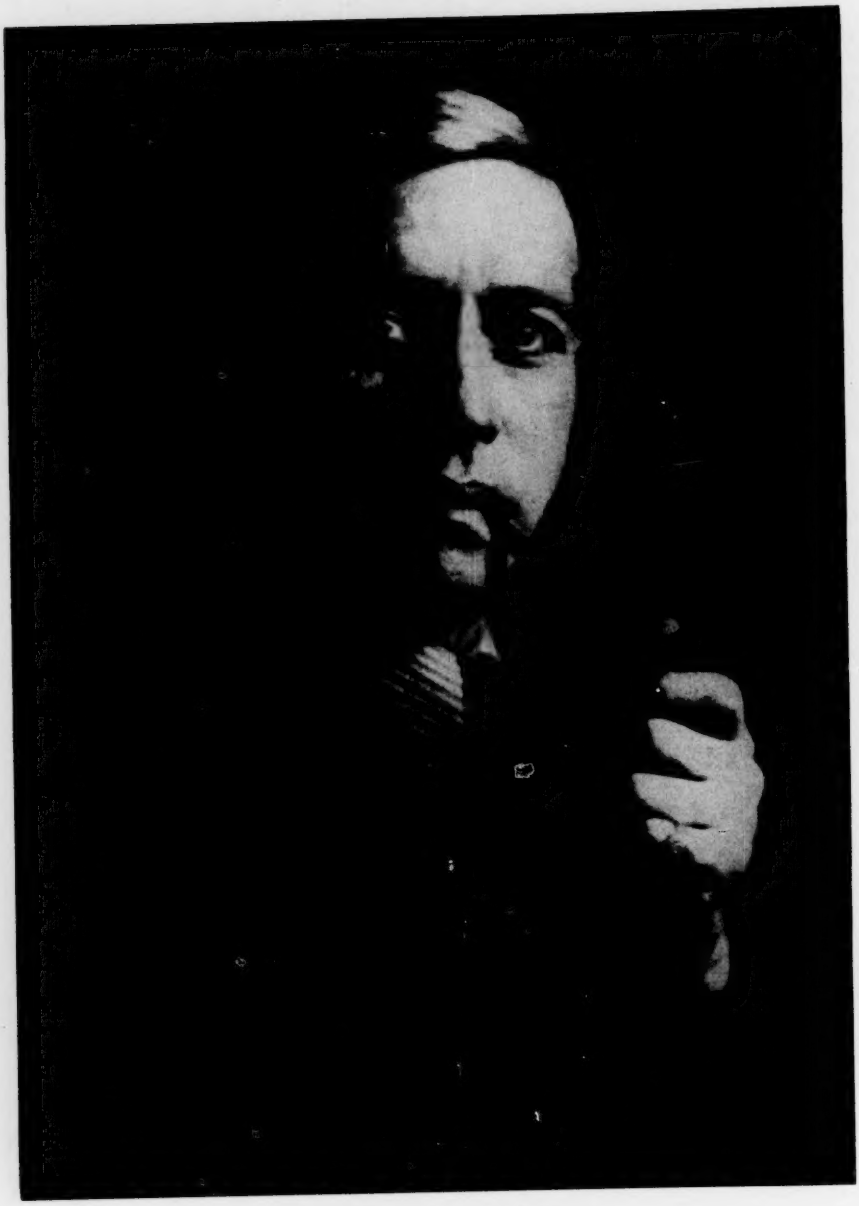

CHARLES LEONARD HUSKINS

CHARLES LEONARD HUSKINS

1897-1953

THE premature death of Charles Leonard Huskins, Professor of Botany at the University of Wisconsin, on July 26, 1953, brought to an untimely end a distinguished scientific career. To those of us who were associated with him over a considerable segment of his professional career it appeared that he had reached a point in his own personal development and experience where he was about to realize the true value of his many undoubted talents. His wide interests, retentive memory, vivid personality and not inconsiderable oratorical ability made him an outstanding spokesman for science in Education and Philosophy.

Professor Huskins was born in Walsall, England, but at the age of ten he emigrated with his parents to Alberta, Canada. He received his bachelor's and master's degrees from the University of Alberta and won the "1851 overseas Scholarship" which enabled him to study further at the University of London from which institution he received the Ph.D. in 1927 and the D.Sc. degree in 1934. From 1927-1930 he worked with the late William Bateson at the John Innes Horticultural Institution in England. In 1930 he was invited to join the faculty of McGill University in Montreal as associate professor of Botany. His deep conviction that Genetics was a biological science and should be separated from, though integrated with, both Botany and Zoology led to the establishment of Canada's first Department of Genetics at McGill in 1934 with Professor Huskins as chairman. This infant department flourished under his direction and rapidly became one of the most active such research centers in the world. In 1945 Professor Huskins went to the University of Wisconsin as Professor of Botany where he soon established a semi-autonomous section devoted to experimental cytology.

Professor Huskins was internationally renowned and was frequently invited to lecture and work in many parts of the world including Germany, Russia and France. He was a visiting professor at the University of California in 1938 and a Guggenheim Fellow at Columbia in 1942. He was an active member of many professional societies including the Royal Society of Canada, the Medical and Biological section of which he was president in 1951.

In 1923 he married Margaret Villy, a native of Manchester, England and at the time, a lecturer in English at the University of Alberta. Mrs. Huskins was a charming, intelligent and kindly woman who was never known to utter a harsh word concerning anyone. Her death some six months before that of her husband was a blow which undoubtedly hastened Professor Huskins' demise. They are survived by three children: Sheila, Olwen and John.

The impact of the news of his death on his associates and students is probably best expressed by quoting excerpts from a number of letters which we have received from various people who worked with him at various times.

A. W. S. HUNTER, Central Experimental Farm, Ottawa: "I was one of his first graduate students at McGill. I started my post-graduate work . . . almost coincident with the appearance of Darlington's "Recent Advances in Cytology." Huskins took exception to so many points in this work that there was not enough room in the margins for all the notes and comments he wanted to make, so he sent his copy up to the bindery and had it completely interleafed with blank pages. At that time Huskins was feuding with the medical faculty, attempting to convince them of the value of including some instruction in genetics in the medical course." [His hope has now been realized.] "As a man Huskins was always good to his students. He was very out-spoken and often sarcastic when he tangled with a member of another department whose views did not agree with current genetic principles, but with us he was always patient and usually jolly and full of good humour."

SHELDON REED, Dight Institute, University of Minnesota: "I was flattered to be offered

my first full-time university job with such a vigorous and able scientist. The primitive facilities did not seem to retard the work or enthusiasm of an excellent group of graduate students. They were both stimulated and encouraged by the 'Chief' to an unusual degree. He included them in his personal life to a point which was practically exhausting. The years with 'Len' at McGill were very happy years. I was free to do my research and teacning as I pleased. He believed vehemently in science in general and genetics in particular. 'Len' once told me that he did not want to rust away—that was the way things worked out."

ROBERT DUNCAN, University of Wisconsin: "I first met C. L. Huskins at breakfast in a dining hall of a University dormitory converted to the use of the members of the Seventh International Congress of Genetics at Edinburgh in 1938. His publications on cytogenetics, chromosome mechanics and structure had impressed me with the author's imaginative approach to the problem. They had not prepared me, however, for the impact of his personality. He was buoyant, interested in others' research activities, a student of world affairs and an arresting conversationalist. One got the impression, as many have remarked, that here was a person of superior qualities.

After Prof. Huskins came to Wisconsin I had the opportunity to know him better. The qualities which first impressed me never dimmed. I observed that both graduate and undergraduate students at Wisconsin found him a stimulating counselor and a wise and kindly confidant. Graduate students admired his enthusiasm for research and perhaps had their first contact with a philosophical basis of scientific methodology in his seminars. They recognized his distrust for blind acceptance of authority and his insistence on a simple statement of a problem, the use of appropriate materials and methods and the constant need for checking the validity of conclusions."

MARY E. SANDERS, University of Wisconsin: "My acquaintance with Dr. Huskins began with an interview at Wisconsin during which I was impressed and surprised by his straightforward manner. He arranged that I should see and be told the disadvantages as well as the advantages of the position, and asked directly for information which most people would have sought indirectly. His readiness to meet and deal with problems directly established a free and open relationship with his associates. His own enthusiasm extended to those around him with such energy that people were either inspired to agree or else felt compelled to defend their own opposing opinions. The broad range of his interests found expression in his contributions to Wisconsin's program of Integrated Liberal Studies. He served on the committee which drew up the plans for the program and was one of the professors in charge of the biology course. He was deeply concerned with the necessity for the layman to understand the attitudes, limitations and possibilities of science and therefore, concerned with the kind of emphasis given to courses for non-science students."

GEORGE SETTERFIELD, University of Wisconsin: "Knowing Dr. C. L. Huskins during only the last two years of his eventful life, I must present a limited picture of this many faceted man. He left the undeniable impression of being a highly civilized person endowed with great powers of leadership. His weaknesses arose primarily not from lacks but rather from excesses of the very characteristics which made him exceptional. His vigorous example moved many of us to levels of thought and action which we would never have reached independently."

Many others, especially those who were close to Len over a considerable period, became inarticulate when asked for comment. The reason is summed up by Mrs. Norah Stewart Allen, a former graduate student at Wisconsin: "One's feelings on the loss of a teacher who has also become a good friend and adviser are most difficult to express by the written word."

Leonard Huskins was a complex and vivid personality; an idealist and humanist who fought tenaciously for what he believed to be right. His finest monument will be in the work of those whom he inspired and stimulated.

G. B. WILSON
AND
MERTON LOVE

Michigan State College
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THE DILEMMA OF THE Rh-Hr BLOOD TYPES

FROM 333 published items from 1929 to date, which required 17 pages for their listing, the author selected for this volume* about 80 of his papers "as his most representative and important contributions to this subject, thus giving an idea of how knowledge in this field developed." The dangers of repetition of which the author was aware "have been avoided by careful selection" and "the insertion of occasional explanatory editorial notes also helps to bring the subject matter up to date." After a careful study of this volume, it is clear that the author did not quite achieve his goal. This is most unfortunate because the important discoveries of this obviously talented author are diluted and even lost by ill-advised insertion of some papers which contain inaccuracies not corrected by editorial notes and others, filled with fanciful speculation not supported by critical experimentation and analysis, e.g. the agglutinogen C of the ABO system, autoantibodies in physiological jaundice, valencies of Rh antibodies, analogies to allergic phenomena, and other items mentioned below.

The historically important 1940 and 1941 papers with Landsteiner form Chapters 3 and 5 respectively, but the editorial comment on page 52 is contradicted on page 46 of the text, and there is a conflict of dates on page 24 and the first sentence of the preface. The diagrammatic representation of the two varieties of Rh antibodies shown on page 68 is given six other times, and in general there is much overlapping of the same subject matter in many papers.

All workers in the field question the wisdom of employing in Rh work the term "conglutinin" which does not correspond to the definition given by Landsteiner and others. There is no evidence whatever to justify invoking the X-protein and to claim its identity with "conglutinin" because the same effect can be produced by dextran, PVP, and other high-molecular colloidal substances. On pages 190 and 210 the claim is made that his "albumin-plasma" method is more sensitive than the antiglobin technique, but there is no warning or editorial comment to inform the reader that subsequently (p. 708) the author has changed his mind regarding the relative sensitivity of these two tests. There are a number of chapters on prognosis and the indications for therapy based exclusively on titers of maternal serum, but there is no reference to the excellent studies of Mollison and Pickles on the importance of hemoglobin, bilirubin, and reticulocyte counts in the affected infant's blood.

The source for much of the data in Table 4 on page 127 duplicated on page 640, particularly those dealing with physico-chemical properties (sedimentation rate, probable molecular weight) of "univalent" and "bivalent" antibodies is not given, and actually these determinations for Rh antibodies have not yet been made. Although his papers on the pathogenesis of kernicterus are not included, he apparently still adheres to his hypothesis of intravascular agglutination for which there is not a shred of evidence.

Wiener's rigid and fixed views on the heredity of the Rh-Hr factors are well known and will be referred to in the review of his *Rh-Hr Syllabus*. Suffice it to state here that (1) he fails to take into account varying degrees of linkage as amply demonstrated in experimental genetics and (2) he is too deeply committed to view this subject objectively. In short, Wiener has taken personal possession of the multiple allele theory and without benefit of original data and has extended it to include the MNSs factors and with it an entirely new terminology. One may also question the propriety of the satire expressed in the title of chapters 36, 37, and 41, i.e. ". . . a C-D-E Fable," "More C-D-E Nonsense," etc.

Among the more stimulating chapters, aside from the excellent papers with Landsteiner, one may include those dealing with the mosaic structure of agglutinogens, the origin of naturally occurring antibodies, the half-life of antibodies and chapter 83 on the quantitative Coombs test, but here again the effect is marred by the author's penchant to create new terminologies which are not apt to survive and a tendency to oversimplify. It is evident that the reader whether a tyro or a learned scholar will be lost in the genetics and other aspects of the Rh-Hr factors, unless the subject be presented more briefly and far more objectively in a medium other than a voluminous collection of insufficiently edited reprints of papers. On the other hand, the hematologist, serologist, geneticist, and particularly the immunochemist who cannot study Wiener's numerous publications as they appear, may find it convenient to have a sampling in one volume.

One cannot help but gain the impression that this remarkably enthusiastic author brought all the forces at his disposal toward a too rapid solution of all problems associated with the serology, genetics, and clinical aspects of the Rh-Hr blood factors.

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**Alexander S. Wiener.* Rh-Hr Blood Types. Applications in Clinical and Legal Medicine and Anthropology. i-xvi, 763 pp. Grune and Stratton, New York. 1954. $11.50.

AN Rh-Hr SYLLABUS

THIS brief monograph* would be far more useful and representative if the author were somewhat cognizant and tolerant of the views of other workers who also made notable contributions to the subject. Wiener's violent rejection of the concept of linkage and his hypersensitivity to the CDE nomenclature is unfortunate because in the final analysis multiple alleles cannot be easily differentiated from closely linked genes (pseudoalleles). And so, the results of all family and racial studies are compatible with either theory in spite of definitions in standard medical dictionaries very likely taken from, or contributed by, Wiener himself. Faced with these facts and the knowledge that in several instances (*Drosophila*, maize), multiple alleles gave way to linkage with a very low cross-over rate, geneticists conducting courses in universities and medical schools invariably prefer to teach in terms of separate loci, D-d, C-c, E-e.

The author's failure to recognize the commonly accepted "D^u" and his preference for the Germanic "R" for Rh variants does not make it easier for the medical secretary or the printer, and its oral expression is very difficult. The only motive for the re-introduction of C as a factor common to A and B and the unwarranted change of Fy^a (Duffy) to F is to embarrass the DCE nomenclature which nevertheless will continue to thrive in spite of the author's humorous and rather undignified tomfoolery on pages 28 and 29. There is only a personal whim to justify the change from Jk^a (Kidd) to J. The terms Fy^a-Fy^b and Jk^a-Jk^b were introduced by their discoverers for the very good reason that their genes as well as the genes for the factors D-d, C-c, E-e, K-k, and S-s were shown to have equal dominance.

All his unproven hypothesis regarding the pathogenesis of kernicterus, physiological icterus, hemorrhagic manifestations in hemolytic disease, conglutinin valencies, molecular size, and chemical properties of Rh antibodies are passed off as established or almost established facts. One may well question the author's view on page 41 regarding the intragroup isoimmunization in the 10 (?) percent due to blood factors other than D (Rh$_o$). Now that a panel of selected test cells is available, hospital laboratories report an ever increasing success in the identification of these rare antibodies. Furthermore, the disease in the infant may be just as severe as in the typical Rh cases. His remarks on page 53 regarding "inexperienced operators" preferring the umbilical vein for replacement transfusion are highly prejudicial and unrealistic since the umbilical route happens to be the method of choice on the part of the vast majority of rather skilled workers.

The author does not fully appreciate the importance of cord hemoglobin and bilirubin values (Mollison) and reticulocyte counts (Pickles) as criteria for replacement therapy, and he places far too much faith on maternal titers. In his description of the therapy of hemolytic transfusion reaction, the author fails to mention that many patients who survive the anuric stage die later because electrolytes lost in the polyuric stage are not replaced.

The several tables on the heredity of Rh (DCE) and Hr (dce) factors and their gene (or chromosome) distribution in the several races are most useful and instructive provided that the reader (1) will be able to convert to the DCE equivalents, and (2) will keep an open mind on the ultimate genetic theory. In cases of disputed paternity, experience has shown that for court officials and lawyers exclusion or no exclusion is more simply expressed in terms of the DCE terminology.

If the objective of the author was to prepare the reader for his longer volume *Rh-Hr Blood Types*, he has indeed succeeded. But what will the author reply to those who have occasion to consult the publications and books of workers other than Wiener, only to find a wide gap in terminologies? His success with this booklet would have been assured if the author suppressed his tendency to coin new terms and relegated his numerous speculative concepts to footnotes. These considerations detract from the value of this booklet which contains much useful and concise information on genetic and serologic definitions, serology of Rh-Hr types, hemolytic disease of the newborn, therapy by replacement transfusion, autoimmunization, classifications of human population in terms of gene frequencies and medicolegal applications. On the whole, this booklet may be accepted as a minority report.

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**Alexander S. Wiener.* An Rh-Hr Syllabus. 82 pp. Grune and Stratton. New York. 1954. $3.75.

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